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THERAPY OF NERVE DEAFNESS AND TINNITUS AURIUM

USE OF LARGE DOSES OF THIAMINE HYDROCHLORIDE AND EVALUATION OF RESULTS, WITH A SOURCE OF POSSIBLE ERROR IN INTERPRETATION OF IMPROVEMENT

GEORGE E. SHAMBAUGH JR., M.D.
CHICAGO
AND
MILTON L. JENNES, M.D.
WATERBURY, CONN.

Among the more recent approaches to the therapy of eighth nerve disorders is that of the administration of vitamins, more specifically the use of the various factors of the vitamin B complex. Of these, vitamin B_1, or thiamine hydrochloride, by virtue of being considered the "antineuritic" vitamin, has received the greatest attention both in the medical and in the lay press.

In ascertaining the efficacy of any treatment, scientific control of the results is essential. A series of cases of deafness comparable without treatment may serve as the control for the series of cases with treatment. If the defect is bilateral and only one ear is treated, as in surgical operation for deafness, the untreated ear furnishes the best control. In a case of long-standing chronic deafness, the previous course may in itself be taken as the control period.

In the treatment of deafness, it is necessary to recognize the considerable psychic element in the response of deaf persons to any new therapy. Because of the increased effort required to understand what is said, a deafened person easily falls into the habit of inattention. Stimulated by hope through any new treatment, he begins to pay more attention and therefore hears more, though his hearing remains unchanged, as shown by objective tests, for these always require the full attention of the patient.

Concerning objective tests of hearing Holmgren [1] stated:

> . . . the whispering distance and the conversational distance even if recorded by the most experienced and trained examiner and under the most favorable con-

1. Holmgren, G., in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1939, p. 410.

ditions have only a limited value and must under no consideration be taken as a sure gauge of the patient's hearing and as such used for comparison with values obtained at another examination. . . . The best method of obtaining objective and commensurable values is to make use of an audiometer.

Caution must, however, be observed in the use of audiometric examinations for proof of hearing improvement. Audiograms taken over a length of time in any case of deafness without therapy may show a relatively wide fluctuation of hearing. Two factors may be responsible for this. It is a common observation that fluctuation in hearing within certain limits occurs in most cases of deafness. The factors contributing to this are many: fatigue, atmospheric pressure, temperature, time of day, intercurrent infections of the upper respiratory tract and other factors both known and unknown. Then there is the error inherent in audiometers, usually within a range of 10 decibels, which may show up in repeated audiograms made in the course of one morning.

Bunch [2] has presented the results of repeated audiometric testing over long periods in cases of otosclerosis. In a case in which two tests taken six years apart showed no appreciable progress of the disease, fifty-seven other tests taken in the intervening period showed considerable variation; the vast majority of the recordings lay within a range of 10 or, at the most, 15 decibels, but in some frequencies there was a variation of as much as 20 to 25 decibels. And here the examiner and the audiometer were constant.

Audiograms taken months apart and showing slight improvement should therefore not be accepted by the critical otologist as proof of hearing improvement. Repeated audiograms made before a course of treatment and again at its completion should be required for judging improvement of hearing. In cases in which treatment has been carried on over a long period, a series of audiograms made at intervals of perhaps a month should be acceptable, provided they show a sustained improvement of more than 10 decibels.

It should go without saying that no particular conclusions can be drawn from the results of therapy in cases of deafness due to self-limited conditions, in which the tendency is toward recovery with no treatment. Yet the recent furor over the use of prostigmine originated because of just such an observation [3]: A considerable number of patients with acute blocking of the eustachian tube improved under prostigmine therapy. The authors then concluded that prostigmine had been responsible for the cures despite the fact that inflation, which usually clears up such a condition, was used.

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2. Bunch, C.: The Progress of Deafness in Clinical Otosclerosis, Laryngoscope **49**:793 (Sept.) 1939.

3. Davis, T., and Rommel, J.: Treatment of Deafness and Contiguous Nervous Disorders with Prostigmine, Arch. Otolaryng. **29**:751 (May) 1939.

It is also possible for errors to occur, in the appraisal of therapeutic measures, through faulty diagnosis. In 1938 one of us [4] called attention to the loss of hearing for high tones in cases of secretory otitis media.

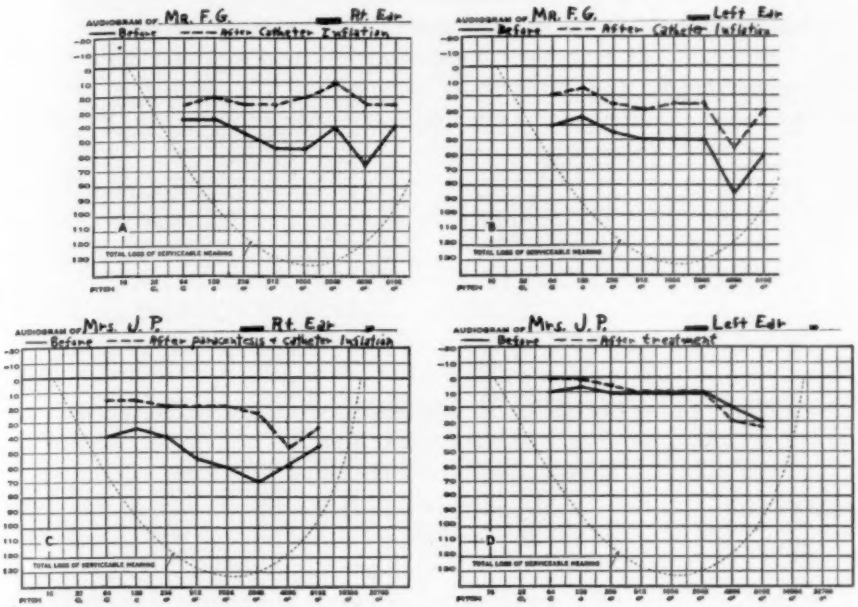

Fig. 1.—*A* and *B*, bilateral secretory otitis media, with loss of hearing for both high and low tones. *C* and *D*, unilateral secretory otitis media, with loss of hearing for both high and low tones.

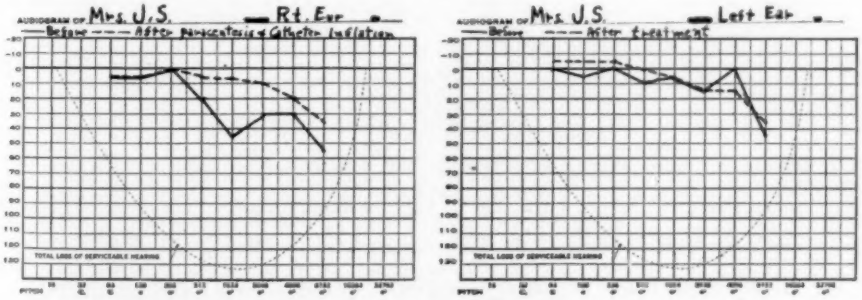

Fig. 2.—Unilateral secretory otitis media, with loss of high tones only simulating nerve deafness.

It was demonstrated that the loss for the high tones could be as great as that for the low tones (fig. 1). Routine audiometric and tuning fork testing of the patients has further disclosed that many retain normal

4. Shambaugh, G. E., Jr.: Recent Advances in the Diagnosis and Treatment of Deafness, Ann. Otol., Rhin. & Laryng. **47**:636 (Sept.) 1938.

hearing for the low tones and show a loss only in the higher tones (fig. 2). The audiogram simulates that of primary nerve deafness, and the Rinne test for 256 double vibrations may yield a positive or a shortened positive reaction. Examination of the ear may show a tympanic membrane normal in all respects except for the easily overlooked pale

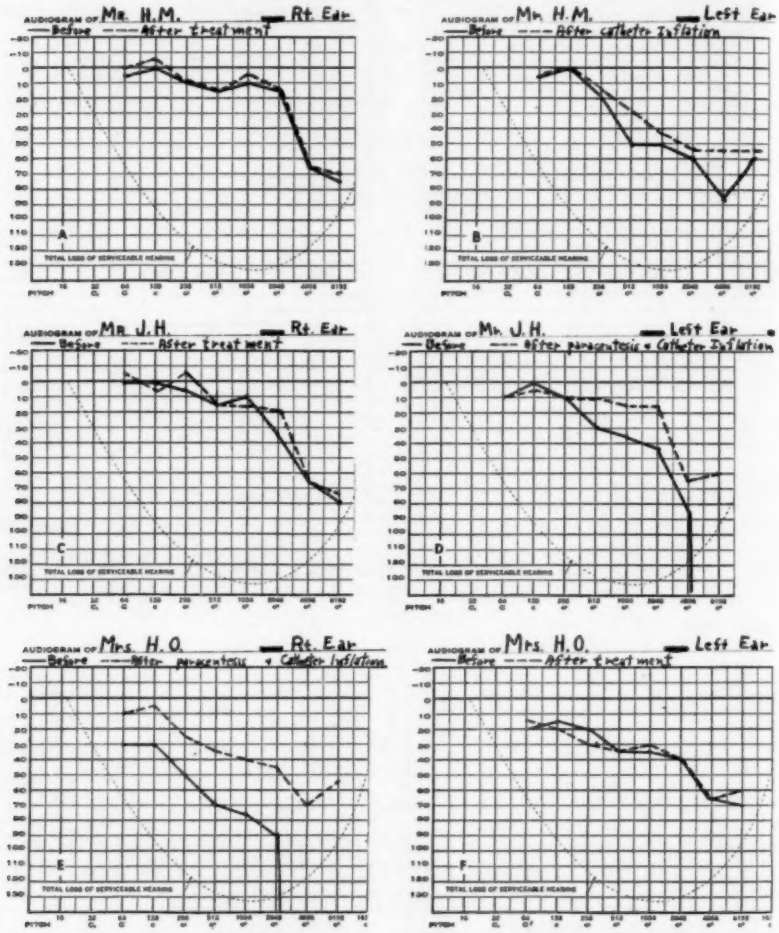

Fig. 3.—Audiograms in 3 cases of unilateral secretory otitis media superimposed on high tone nerve deafness. In the case represented by *C* and *D*, two paracenteses and twelve catheter inflations were required before the transudate in the middle ear was completely eliminated.

yellow or amber color due to the transudate within the tympanum. Unless the tympanum is only partially filled, a fluid level line, or *niveau*, is not present. It is still easier to overlook secretory otitis media when the drum membrane is thickened and the characteristic yellowish color is not visible.

Secretory otitis media superimposed on already established nerve deafness is another source of error in diagnosis (fig. 3). With tinnitus and deafness already present, the increased deafness may be attributed to progression of the perceptive deafness.

Since secretory otitis media tends to clear up spontaneously and the functional tests suggest nerve deafness and since the diagnosis could easily be missed, it would be easy to conclude that any given form of therapy in such a case had resulted in improvement in a case of nerve deafness. Familiarity with the characteristic yellowish color of the transudate shining through the drum membrane and routine use of diagnostic catheter inflation with the auscultation tube should minimize this source of error.

Two recent articles on the use of prostigmine methylsulfate in deafness, by Houser, Campbell and Schluederberg [5] and by Ersner, Rush and Myers,[6] are commendable for the care shown by these writers in the diagnosis of conditions and the evaluation of therapy in their cases.

A review of the literature on the use of thiamine hydrochloride in the treatment of deafness and tinnitus aurium shows that comparatively few articles on the subject have been published and that some of the studies on which these were based were carried out with inadequate controls. The stimulus to the use of vitamin B in otologic practice was provided in 1939 by Selfridge,[7] who two years previously had discussed deafness from a nutritional standpoint.[8] He used nicotinic acid as well as thiamine hydrochloride, claiming for each a measure of success. The recorded audiograms, taken at intervals of many months, showed some improvement in the hearing, most marked in the lower tones (128 to 512 double vibrations).

Veasey [9] used vitamin B_1 and the vitamin B complex, singly or in combination, in 7 cases of deafness and tinnitus which included cases of otosclerosis as well as cases of nerve deafness. In 1 case slight improvement of hearing was shown by the tuning fork test; in a second

5. Houser, K.; Campbell, E., and Schluederberg, H.: The Treatment of Chronic Deafness and Tinnitus Aurium with Prostigmine, J. A. M. A. **115**:994 (Sept. 21) 1940.

6. Ersner, M.; Rush, I., and Myers, D.: Prostigmine Methylsulfate in the Treatment of Deafness and Tinnitus Aurium: Report of Fifty-Nine Cases with Negative Results, Arch. Otolaryng. **33**:193 (Feb.) 1941.

7. Selfridge, G.: Nicotinic Acid and the Eighth Nerve, Ann. Otol., Rhin. & Laryng. **48**:39 (March) 1939; The Eighth Nerve in Relation to Thiamine Chloride and Nicotinic Acid, ibid. **48**:419 (June) 1939; Eighth Nerve Deafness from a Nutritional Standpoint, ibid. **48**:608 (Sept.) 1939.

8. Selfridge, G.: Chronic Progressive Deafness from a Nutritional Standpoint, Ann. Otol., Rhin. & Laryng. **46**:875 (Sept.) 1937.

9. Veasey, C. A., Jr.: Nature of Vitamin B and Its Components with Special Reference to Nerve Deafness, Arch. Otolaryng. **31**:74 (Jan.) 1940.

case there was slight improvement in one ear, with an equivalent loss in the other; in the other cases there was no change.

In an interesting case reported by Brandenburg,[10] severe bilateral tinnitus and deafness following extensive radiation therapy for carcinoma of the uterine cervix were relieved completely by ten daily intravenous injections of 10 mg. of thiamine hydrochloride. After two days the dose was increased to 15 mg. The apparent cause of the aural symptoms, which were present for three weeks before institution of treatment with thiamine hydrochloride, was toxic absorption following the destruction produced by the extensive irradiation of tissue. In contemplating the outcome of the case the author stated: ". . . it is not impossible that the normal recovery from the radiation sickness might have played some part in the picture."

Childrey,[11] in a well controlled study, administered thiamine hydrochloride as well as the entire vitamin B complex to many patients who had nerve deafness or mixed deafness with a large neural element, and audiograms of 26 patients so treated showed no gain in hearing. He used thiamine hydrochloride in many cases of tinnitus, and in only a single case did it appear that the use of the drug gave any relief. In that case the tinnitus was associated with the menopause. In his cases the thiamine hydrochloride was given subcutaneously in doses of 3,000 to 5,000 international (U. S. P. XI) units or orally in an average daily dose of 672 international (U. S. P. XI) units.

Childrey presents the reasonable query: If nerve deafness is to be explained by a deficiency of vitamin B, why, then, is there no other evidence of avitaminosis, and why should such deficiencies appear to predominate in older patients?

It seems to us that since well developed deficiency states such as beriberi, scurvy and pellagra are not known to be associated with loss of hearing, it is unlikely that subclinical deficiencies produce nerve deafness. Furthermore, human subjects in whom a deficiency of thiamine hydrochloride was induced[12] did not show symptoms referable to the eighth nerve.

PERSONAL OBSERVATIONS

Five patients with progressive primary nerve deafness of long standing with marked tinnitus were placed on a therapeutic regimen of daily (except Sunday) intravenous injections of 50 mg. (16,650 international [U. S. P. XI] units) of thiamine hydrochloride over a period of two

10. Brandenburg, K.: Relief of Neuritis of Eighth Cranial Nerve with Vitamin B_1, Arch. Otolaryng. **31**:189 (Jan.) 1940.

11. Childrey, J.: Vitamin B and Other Measures in the Treatment of Deafness, Laryngoscope **50**:648 (July) 1940.

12. Williams, R.; Mason, H.; Wilder, R., and Smith, B.: Observations on Induced Thiamine (Vitamin B_1) Deficiency in Man, Arch. Int. Med. **66**:785 (Oct.) 1940.

weeks. One patient (case 5) failed to complete the course of treatments after he received nine injections without any relief of symptoms. Two of these patients (cases 1 and 5) had previously been given intramuscular injections of thiamine hydrochloride in 10 mg. doses without benefit.

Not one of the 5 patients experienced any subjective or objective improvement in the hearing. All had audiometric tests before and after treatment, without any demonstrable improvement. In no case was there any relief of the tinnitus after treatment. In 1 instance (case 4) there was temporary complete disappearance of the tinnitus after the fifth injection, but this lasted only a few days. In another instance (case 5), during the course of treatment the tinnitus, which had previously been intermittent, became constant and more severe. The

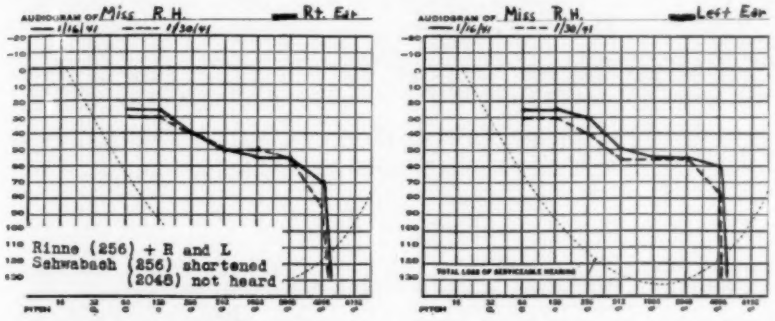

Fig. 4 (case 1).—The straight line indicates the hearing before treatment with thiamine hydrochloride; the dash line, the hearing after daily intravenous injections of 50 mg. of thiamine hydrochloride.

wife of 1 patient (case 4) thought that his hearing had improved, but this opinion was not substantiated audiometrically.

Previously, one of us (G. E. S. Jr.) had employed thiamine hydrochloride sporadically in the treatment of nerve deafness and tinnitus aurium and in smaller doses than that used in this series, with uniformly negative results.

REPORT OF CASES

CASE 1.—Miss R. H. (fig. 4), aged 39, complained of severe ringing and roaring tinnitus of over one year's duration and of deafness, which had been present for many years, worse in the past year. The use of sedatives and the removal of foci of infection gave no relief from the aural symptoms. The Kahn reaction of the blood was negative. Between Dec. 26, 1940 and Jan. 11, 1941 she was given five 10 mg. doses of thiamine hydrochloride intramuscularly, and from Jan. 16 to Jan. 29, 1941, 50 mg. of thiamine hydrochloride intravenously daily except two Sundays. There was no subjective lessening of the tinnitus and no subjective or audiometric improvement in the hearing.

CASE 2.—Mr. J. L. (fig. 5), aged 53, complained of roaring tinnitus of two years' duration without subjective deafness, although audiometric testing showed marked nerve deafness. He had had removal of foci of infection without relief. The Kahn reaction of the blood was negative. From Jan. 30 to Feb. 12, 1941 he was given 50 mg. of thiamine hydrochloride intravenously daily except two Sundays. The tinnitus was unchanged, and there was no audiometric improvement in the hearing.

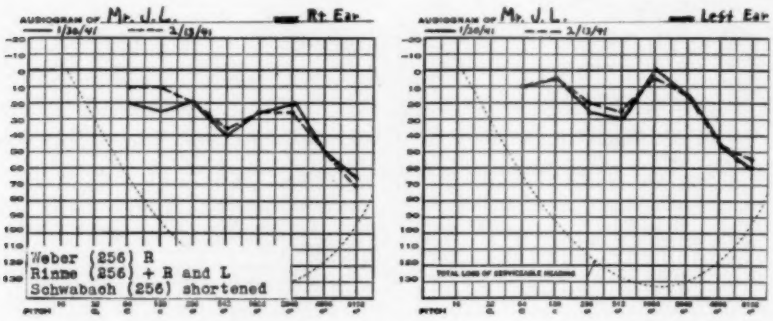

Fig. 5 (case 2).—The straight line indicates the hearing before and the dash line the hearing after treatment with thiamine hydrochloride.

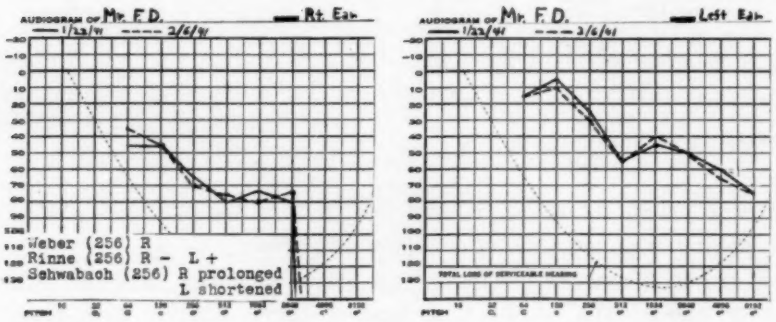

Fig. 6 (case 3).—The straight line indicates the hearing before and the dash line the hearing after treatment with thiamine hydrochloride.

CASE 3.—Mr. F. D. (fig. 6), aged 70, had complained of deafness in the right ear for thirty-five years and in the left ear for five years, with continual tinnitus in the right ear for twenty-five years and in the left ear for one year. Hearing tests showed bilateral nerve deafness, superimposed on an old unilateral conductive defect in the right ear. Roentgenograms of the teeth and the Kahn test of the blood revealed nothing abnormal. From Jan. 24 to Feb. 6, 1941 he was given 50 mg. of thiamine hydrochloride intravenously daily except two Sundays. There was no change in the tinnitus and neither subjective nor audiometric change in the hearing.

CASE 4.—Mr. J. R. (fig. 7), aged 62, had complained of bilateral deafness with tinnitus for eight years. Roentgenograms of the teeth and the Kahn test of the blood revealed nothing of importance. From Jan. 16 to Jan. 29, 1941 he was given 50 mg. of thiamine hydrochloride intravenously daily except two Sundays. After the fifth injection he noticed a temporary complete disappearance of the tinnitus for a few days. At the completion of treatment the tinnitus was as bad as ever, and there was neither subjective nor audiometric improvement in the hearing, although the patient's wife thought his hearing had improved.

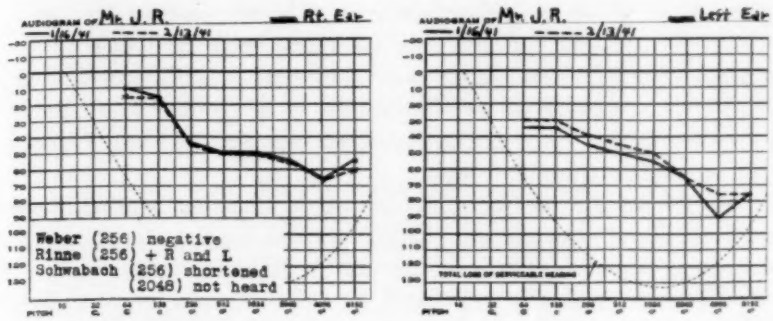

Fig. 7 (case 4).—The straight line indicates the hearing before and the dash line the hearing after treatment with thiamine hydrochloride.

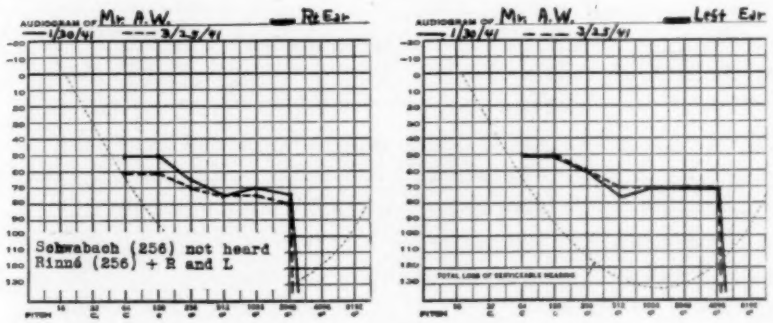

Fig. 8 (case 5).—The straight line indicates the hearing before and the dash line the hearing after treatment with thiamine hydrochloride.

CASE 5.—Mr. A. W. (fig. 8), aged 50, had complained of increasing deafness for sixteen years, with severe tinnitus. Several abscessed teeth were removed without producing any effect on the tinnitus. On Dec. 20, 1940 the patient was given 10 mg. of thiamine hydrochloride intramuscularly, and on Jan. 30, 1941 daily intravenous administration of 50 mg. of thiamine hydrochloride was begun, but after nine injections the patient stopped coming for treatment. When he returned for audiometric testing, on March 25, he said that he had stopped coming for injections because the tinnitus, which had previously been intermittent, had, during

the course of treatment, become continual. The deafness was not changed subjectively or audiometrically.

SUMMARY

The evaluation of therapy for the improvement of hearing in deafened persons must be based on repeated audiometric tests. To be significant there must be a sustained improvement of at least 10 decibels above the pretreatment level.

Secretory otitis media may be easily overlooked and the condition mistakenly diagnosed as nerve deafness. The response to therapy may therefore be wrongly interpreted as an improvement in a case of nerve deafness. This error can occur even more readily when secretory otitis media is superimposed on already established nerve deafness. Some of the improvement in nerve deafness with therapy reported in the literature might be explained on this basis.

A review of the literature on the use of thiamine hydrochloride in the alleviation of deafness and tinnitus aurium suggests that there is no conclusive evidence in favor of the use of thiamine hydrochloride for this purpose. This is substantiated by the uniformly negative results obtained by us in a series of cases of nerve deafness and tinnitus aurium in which large daily doses of thiamine hydrochloride were administered intravenously over a period of two weeks.

122 South Michigan Avenue, Chicago.
18 Crescent Street, Waterbury, Conn.

CHEMOTHERAPY IN ACUTE NONDIPHTHERITIC LARYNGOTRACHEOBRONCHITIS

HERBERT GRAEBNER, M.D.
NEW YORK

The evaluation of the clinical results obtained by the use of chemotherapeutic agents is being constantly recorded in present day literature by numerous investigators. The use of chemotherapeutic measures in the routine treatment of nondiphtheritic croup has been in effect at Willard Parker Hospital [1] during the past eighteen months, and the results noted following this procedure are hereby presented.

TABLE 1.—*Mortality Rates in Various Classifications of the Two Groups (One Without and One With Chemotherapy)*

	Group 1 1937-1938	Group 2 1939-1940
Total admissions	217	241
Deaths	23	16
Mortality (%)	10.5	6.5
Intubations	34	17
Deaths	12	3
Mortality (%)	35.8	17.6
Tracheotomies	28	43
Deaths	7	11
Mortality (%)	25	25.8
Cases of pneumonia	29	29
Percentage of total admissions	13.3	12
Deaths	12	7
Mortality (%)	41.3	24

In order to arrive at a fairly accurate conclusion relative to the effectiveness of these agents, a comparison was made of the statistics derived from the cases of nonspecific croup prior to the inauguration of chemotherapy with those from an approximately equal number of cases in which this treatment was routinely employed. Inasmuch as there had been no remarkable variation in the general management of the patients in the croup wards in the two series, it was felt that any change in the mortality rate may reasonably be assumed to have been proximately the result of the chemotherapy employed.

STATISTICS

Group 1.—During 1937 and 1938 a total of 217 patients were admitted to the croup wards of the hospital. There were 23 deaths in this group—a mortality rate of 10.5 per cent. A further classification

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

1. From the records of Willard Parker hospital.

of the patients in whom intubation was necessary in addition to the usual palliative treatment shows 34, with 12 deaths—a mortality rate of 35.8 per cent. This percentage indicates in a comparative way the increased severity of the disease due to progress from a mild to a severe type. Further extension of the disease is noted in the group in which tracheotomy was necessary. There were 28 patients in this group, with 7 deaths—a mortality rate of 25 per cent. This percentage again reflects the severity of the infection due to wider involvement of the tracheobronchial tree and adjacent lung substance. The final classification shows the incidence of pneumonia as a complication in the patients of this group. In 29, or 13.3 per cent, of the 217 patients admitted, either bronchopneumonia or lobar pneumonia developed. There were 12 deaths due to this complication—a mortality rate of 41.3 per cent.

Group 2.—During 1939 and up to July 1, 1940 a total of 241 patients were admitted to the service, of whom 16 (6.5 per cent) died. In this group are included some patients with a severe type of disease, for whom chemotherapy was routinely used in addition to the general procedure followed in the treatment of the patients in group 1. The comparison of the mortality rates of the two groups is therefore of interest. Among the 17 patients for whom intubation was indicated and chemotherapy was used, there were 3 deaths, a mortality rate of 17.6 per cent. When this is compared with the percentage in this class of group 1, a marked decline is noted. In all the patients in whom tracheotomy was performed, chemotherapy was employed as a routine measure. In this class, with severe involvement of the respiratory tract, always associated with a high mortality rate, the use of chemotherapeutic agents seems to have been least effective. There were 43 patients, with 11 deaths—a mortality rate of 25.8 per cent. Complicating pneumonia developed in 29 patients, or 12 per cent of the total admitted. There were 7 deaths—a mortality rate of 24 per cent. In comparison with this class in group 1 there is a slight increase in the incidence of this complication, with a marked decrease in the mortality rate when the disease did occur.

BACTERIOLOGY

Bacteriologic studies were made in all cases by direct swab of the larynx and subglottic tissues through the laryngoscope and repeated at intervals when invasion of the tracheobronchial tree indicated continued progress of the disease. The laboratory reports on the cultures made are in accord with the findings of numerous investigators in that the infection generally was due to a mixture of organisms, with the streptococci predominating. Whether this mixed infection is primary and the direct cause of the disease or a secondary bacterial invasion following an initial infection by an unknown organism has not been determined. Certain

characteristics of the disease indicate that the condition is a specific clinical entity rather than an ordinary infectious process involving the parts. Jackson [2] stated that he found the influenza bacillus in about 5 per cent of his cases and that there was no means of knowing in how many other cases this organism may have been the vanished primary invader. He further stated that most of the cases of the disease commonly called influenza are also primarily or secondarily streptococcic in character. The outstanding characteristic of his bacteriologic reports is that though other organisms were reported early in many instances, the later reports showed in almost all these cases streptococcic predominance. Gittens [3] also found streptococci most often in his cultures, but frequently associated with staphylococci and pneumococci. Experiments are being conducted at present by resident physicians at Willard Parker Hospital by insufflating bronchial discharges into the respiratory tracts of mice. These animals are anesthetized with ether to insure deep implantation and retention of the material in the tracheobronchial tree. A fulminating type of the disease developed in all the animals, and they have rapidly succumbed. Autopsy showed tissue changes similar to those noted in human specimens. The cultures made generally showed mixed growths, with that of the streptococcus group predominating.

The gross percentages of the predominating organisms found in the cases presented here are as follows:

Streptococcus haemolyticus	35.2%
Streptococcus viridans	28.9%
Staphylococcus aureus	28.3%
Staphylococcus albus	7.3%

TREATMENT

All the patients in group 1 were treated according to the routine procedure established in Willard Parker Hospital for patients with acute nondiphtheritic laryngotracheobronchitis.[4] This consists of inspection of the upper part of the respiratory tract by the direct method, to determine the degree of involvement and the necessity of immediate intubation or tracheotomy, to secure a culture of the exudates present and to clear the respiratory tract of obstructive mucus and crusts. The general physical condition and the local parts are checked at frequent intervals. Rest in bed, mild sedation to combat restlessness, and use of steam inhalations when secretions are abundant and difficult to expel are supplemented by topical applications in the form of drops or sprays. Plenty of fluids and

2. Jackson, C., and Jackson, C. L.: Acute Laryngotracheobronchitis, J. A. M. A. **107**:929-932 (Sept. 19) 1936.

3. Gittens, T. R.: Laryngitis and Tracheobronchitis: Reference to Nondiphtheritic Infection, Ann. Otol., Rhin. & Laryng. **45**:1165 (Dec.) 1936.

4. Neffson, A. H., and Wishik, S. M.: Acute Infectious Croup, J. Pediat. **5**:433 (Oct.); 617 (Nov.); 776 (Dec.) 1934.

liquid diet are given during the acute stage, and supportive measures in the form of clysis and transfusions are instituted when there is evidence of toxemia or circulatory collapse. Extension of the infection to subglottic tissues generally requires mechanical intervention to maintain an adequate airway, and intubation or tracheotomy is performed. The selection of the procedure of choice, the determining of the appropriate time for its performance and the general management of the patient may vary somewhat among individual operators, but all in all there seems to be little difference of opinion relative to the proper course to pursue in each case. Recently there has been a higher ratio of tracheotomies to total admissions in Willard Parker Hospital, due to increased sentiment on the part of the hospital authorities and the visiting members of the otolaryngology staff in favor of early elective tracheotomy over late emergency operations. Experience has shown a definite decrease in the incidence of such complications as atelectasis, emphysema, pneumothorax and circulatory failure as a result of the early performance of tracheotomy.

In addition to the procedure outlined, the patients of group 2 received chemotherapy. This form of treatment suggests itself on the finding of a predominance of streptococcus growth on culture, either in pure form or, more frequently, as a mixed infection. In severe infections the bacteriostatic action of chemotherapeutic agents tends to prevent the development of complications and to shorten the course of the disease process. In the milder types of the disease chemotherapy was not used, as most patients do well under palliative treatment alone and its effectiveness therefore cannot be properly evaluated. The method of administration and the quantity of drugs used in individual cases need not be described. Since this treatment procedure has become an established fixture in modern medicine and its usefulness in appropriate disease conditions has been placed on a sound basis, medical literature is replete with reports on the improvements in these agents, the widened applications and the more complete data regarding their effect on body tissues. The recent studies of Bullowa [5] and his associates has done much in amplifying the literature of these advancements. The development by them of a rapid bedside computation for determining the blood concentration [6] has also been of great value in the proper application of this treatment. A detailed account of the procedure followed at Willard Parker Hospital would differ in no way from present established lines. Sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) or sodium

5. Bullowa, J. G. M.; Ratish, H. D.; Davidson, A., and Lebair, C.: The Administration of Sulfapyridine and Its Congeners in Pneumonias, New York State J. Med. **40**:1450 (Oct. 1) 1940.

6. Ratish, H. D., and Bullowa, J. G. M.: Bedside Test for Sulfapyridine, J. Lab. & Clin. Med. **25**:654-657 (March) 1940.

sulfapyridine (the monohydrate sodium salt of 2-[paraaminobenzenesulfonamido]-pyridine) orally administered was used in most cases. Bronchopneumonia or lobar pneumonia is the frequent end result of a progressive infection of the tracheobronchial tree, and it was the anticipation of this complication in severe laryngotracheobronchitis and the desire to prevent its occurrence, if possible, that determined the use of these compounds. There are not sufficient data available on the effectiveness of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) in these cases to warrant comment at this time. In cases in which the infection of the upper airway was becoming marked but physical examination showed comparatively little pulmonary involvement, sulfanilamide was used. Inasmuch as these conditions have a decided tendency to become emergent and fulminating it is the policy at Willard Parker Hospital to establish as rapidly as possible and to maintain as high a blood concentration of the drug as is consistent with the general trend of the disease process and the tolerance of the individual patient.

COMMENT

An analysis of the statistics of record, with particular reference to the mortality rates of both groups of patients, discloses in general a decrease in mortality in the group that received routine chemotherapy. An exception to this finding is noted in the subdivision in which tracheotomy was required. As stated previously, the general management of the patients in both groups was substantially the same, and it is felt that the change in mortality may be attributed in part to the employment of chemotherapeutic agents. In the total group of patients admitted, a drop from 10.5 to 6.5 per cent is shown. In the subgroup in which the disease was more severe and intubation was necessary, a decrease in the mortality rate from 35.8 to 17.6 per cent is noteworthy. Chemotherapy was used in the treatment of all these patients. In the subdivision in which tracheotomy was required, there was no appreciable change in the mortality rate between the two groups. A possible explanation of this apparent failure may lie in the fact that when the disease has progressed to such a point that radical measures are necessary, the general physical resistance is poor, and circulatory failure and damage to body tissues from toxemia produce death. Although all patients in this subdivision of group 2 received chemotherapy, the treatment was apparently of no value when the physical condition of the patient rendered absorption and retention difficult. The patients of group 2 in whom complicating pneumonia developed showed but slight decline in the incidence percentage as compared with those of group 1. The drugs probably do not often prevent this complication, though they do have a profound effect in the event of its occurrence. In this subdivision of group 2 there were 29 patients, among 241 admitted, or 12 per cent. There were 7 deaths in this class, a mortality rate of 24

per cent, which compares very favorably with 41.3 per cent in the same class in group 1. The records show comparatively few complications involving the upper respiratory tract, such as otitis media, mastoiditis, tonsillitis and sinusitis, in these cases. In view of the presence of an acute infection of the nasopharyngeal space, it is striking that such complica-

TABLE 2.—*Data on Cases in Which Chemotherapy Was Used and Death Occurred*

Case	Age	Time in Hospital, Hr.	Organism	Complications	Treatment
1	10 mo.	7	Staph. albus	Moribund on arrival; laryngospasm; rickets	Intubation; oxygen; calcium gluconate; tracheotomy
2	3 yr.	2	No report	Moribund on arrival; circulatory failure	Intubation in ambulance; oxygen; epinephrine
3	2 yr.	36	Staph. aureus	Autoextubation; circulatory failure	Emergency tracheotomy; sulfapyridine
4	10 mo.	35	Staph. aureus	Lobar pneumonia, type not determined; rickets	Oxygen; intubation; tracheotomy; sulfapyridine
5	9 mo.	19	Staph. aureus; Pneumococcus type XIX	Lobar pneumonia	Intubation; tracheotomy; antipneumococcic serum type XIX; oxygen; sulfapyridine
6	9 mo.	32	Str. viridans	Bronchopneumonia; atelectasis; toxemia; autoextubations	Intubation; tracheotomy
7	15 mo.	48	Staph. aureus	Toxic encephalitis; circulatory failure	Intubation; tracheotomy; sulfapyridine
8	4 yr.	26	Staph. albus	Toxemia; circulatory failure	Intubation; tracheotomy; sulfapyridine; oxygen
9	9 mo.	28	Str. viridans	Lobar pneumonia, type not determined; postmortem blood culture showed Staph. aureus; open ductus arteriosus	Sulfapyridine; oxygen
10	2 yr.	48	Str. viridans	Toxemia	Sulfapyridine; citrated blood
11	2 yr.	2	Str. haemolyticus	Bronchopneumonia; toxemia	Intubation; tracheotomy; sulfapyridine; oxygen
12	1 yr.	60	Staph. aureus	Bronchopneumonia; toxemia	Intubation; tracheotomy; sulfapyridine; oxygen; citrated blood
13	2½ yr.	36	Staph. aureus	Bilateral pneumothorax; toxemia	Intubation; tracheotomy; sulfapyridine; citrated blood
14	15 mo.	7	Staph. albus	Bilateral pneumothorax; circulatory failure	Intubation; tracheotomy; sulfapyridine
15	16 mo.	8	Staph. aureus	Circulatory failure	Intubation; tracheotomy; sulfapyridine
16	7 mo.	22	Staph. albus	Bronchopneumonia; acute otitis media, (Str. haemolyticus)	Intubation; oxygen; sulfapyridine

tions did not arise more frequently. In the event of their recurrence, they were a more or less inconspicuous part of the clinical picture. Acute otitis media was most often encountered and occurred in approximately 5 per cent of the patients admitted. All the other complications were present in less than 1 per cent. Because of the comparatively small number of these sequelae, the influence that chemotherapy exerted was not definitely ascertained, but the course of the disease created the general impression that the drugs were of some value in this respect.

Although these data indicate an advance in efficiency of management of such patients, it was deemed proper to make an analysis of the cases in which death occurred though the patients had received the benefit of chemotherapy. The hospital records were reviewed, and some pertinent data are shown in table 2.

The average age of the patients who died despite chemotherapy was 18 months, and the average stay in the hospital was twenty-six hours. The combination of extreme youth with a rapidly progressing infection, shown by the few hours of survival after the onset of the disease, may have been a factor in determining the fatal end result; in only 1 of the 16 cases in which death resulted was Str. haemolyticus shown to be the predominating organism. The patient was dying on arrival at the hospital. Str. viridans was present in 3 cases, Staph. aureus in 7 and Staph. albus in 4. A postmortem blood culture in case 9 showed Staph. aureus, although swabs of the respiratory tract showed Str. viridans. In case 16 culture of material from the trachea yielded Staph. albus, but the complicating acute otitis media was due to Str. haemolyticus. The patient in case 2 was moribund on arrival, and no report of culture was obtained. As in case 1, in which the patient was also moribund on arrival at the hospital, necessary emergency measures prevented the use of chemotherapy. The direct cause of death was practically always toxemia, circulatory failure or pneumonia. Early hospitalization and radical treatment may have forestalled the fulminating trend of the disease in most of these cases.

SUMMARY

The mortality rates in two groups of patients with acute nondiphtheritic laryngotracheobronchitis have been presented. One group of 217 patients received routine general care, another group of 241 received chemotherapy as adjunct treatment.

Among the patients who received chemotherapy there was a decrease in the mortality percentage from that among the patients who did not receive this treatment.

There was a slight decrease in the incidence of complicating pneumonia in the group in which chemotherapy was used. The mortality percentage decreased markedly in those patients in whom this complication did occur.

An analysis of the observations on the patients who died in the group in which chemotherapy was used showed the drugs to be apparently ineffective in the presence of some groups of bacteria.

In the presence of marked physical exhaustion, the activity of the drugs appears to be impaired.

The evidence of record indicates chemotherapy to be of value in suitable cases in which the predominating organism is amenable to the drugs and the physical resistance of the patient has not been too greatly exhausted.

45 Park Avenue.

INTRACRANIAL COMPLICATIONS OF TONSILLAR ORIGIN

WITH SPECIAL REFERENCE TO THEIR INCIDENCE AND PATHOGENESIS

H. J. HARA, M.D.
AND
CYRIL B. COURVILLE, M.D.
LOS ANGELES

Of all the regional areas in the field of otolaryngology, the tonsils are second only to the larynx and esophagus in the rarity with which infections extend into the intracranial space. Nevertheless, such complications do occur and present considerable variation in the routes by which infection reaches the intracranial space and the nature and extent of the intracranial lesion. It is the object of this contribution to review the literature concerned with the problem, to study the pathogenesis and nature of the consequent lesions and to report 2 cases which have come to our attention.

INCIDENCE

In a survey of a series of 25,000 autopsies performed in the Los Angeles General Hospital one of us (Dr. Courville) found only 1 instance of intracranial complication of tonsillar origin, the report of which appears in this paper. In a series of 10,000 cases of tonsillectomy, Leatherwood [1] cited the case of a child in whom purulent meningitis developed but who recovered. Chisolm and Watkins [2] found 8 cases of thrombosis of the cavernous sinus among 50,000 surgical records at Johns Hopkins Hospital, but in not a single instance was the condition of tonsillar origin.

Seelenfreund [3] reviewed the subject of thrombosis of the cavernous sinus and found 134 cases, in 16 of which the thrombosis was caused by inflammation in and around the tonsils. In a study of the case records of 4,344 children whose tonsils and adenoids were removed at a school clinic in England, Keen [4] found 7 cases in which mastoidectomy was

From the Departments of Otolaryngology and Neurology, College of Medical Evangelists, and the Cajal Laboratory of Neuropathology, Los Angeles County Hospital.

1. Leatherwood, T. F.: Complications Following Tonsillectomy, J. Tennessee M. A. **21**:272 (Nov.) 1939.

2. Chisolm, J. J., and Watkins, S. S.: Twelve Cases of Thrombosis of the Cavernous Sinus, Arch. Surg. **1**:483 (Nov.) 1920.

3. Seelenfreund, B.: Die Genese der Kavernosusthrombose, Zentralbl. f. Ohrenh. u. Rhino-Laryng. **27**:119, 1927.

4. Keen, J. A.: Medical and Surgical Complications of Tonsillectomy in Childhood, J. Laryng. & Otol. **47**:1 (Jan.) 1932.

required within from ten days to six weeks after the operation. In 2 of these cases meningitis developed and the child died. The third child died of septicemia thirteen days after the operation.

Nesbitt,[5] in reviewing the postoperative complications among 1,457 cases, recorded 1 instance of meningitis. Death occurred fourteen days after the tonsils and adenoids had been removed. In an exhaustive study of the causes of fatalities after 332 operations on the nose and throat performed by various surgeons in different sections of the United States, Loeb[6] discovered that the following intracranial complications had occurred: meningitis following adenoidectomy, 1 case; thrombosis of the cavernous sinus following tonsillectomy, 3 cases, and following incision for quinsy, 1 case, and cerebral hemorrhage or embolism following tonsillectomy, 7 cases, and following incision for quinsy, 3 cases. The proportion of morbidity to mortality after operation, naturally, differed in the various centers of population.

NATURE OF LESION

Intracranial lesions of tonsillar origin may be infectious or noninfectious. The noninfectious lesions result from (1) occlusion of the cerebral vessels due to embolism and subsequent softening of the brain tissue, (2) rupture of cerebral blood vessels or (3) anesthesia accidents (status asphyxia) and sensitization incident to the operation on the tonsils. By far the greater number of intracranial complications occur, however, as a result of infection extending by some route to the brain from the tonsillar fossa.

PATHWAYS OF INFECTION FROM THE PHARYNX TO THE INTRACRANIAL STRUCTURES

There are four possible avenues by which infection can be carried to the intracranial space: (1) by way of perineural sheaths of the extracranial nerves, (2) by tissue spaces, (3) by arterial channels and (4) by venous channels. The consequent lesion may be the result of invasion by one or any combination of these avenues in a given case. Uffenorde[7] contended that lymphangitis and lymphadenitis constitute the primary process and secondarily involve the vascular channel. This concept was shared by Brunner[8] in a recent contribution, in which

5. Nesbitt, B. E.: Postoperative Complications and Results of Tonsil and Adenoid Operations in Children, Brit. M. J. **2**:509 (Sept. 15) 1934.

6. Loeb, H. W.: Fatalities Following Operations upon Nose and Throat Not Dependent upon Anesthesia: Study of Three Hundred and Thirty-Two Hitherto Unreported Cases, Ann. Otol., Rhin. & Laryng. **31**:273 (June) 1922.

7. Uffenorde, W.: Die Verwicklungen der akuten Halsentzundungen unter besonderer Berücksichtigung der Beteilgung des Spatium parapharyngeum, Ztschr. f. Laryng., Rhin., Otol. **13**:357, 1925.

8. Brunner, H.: Infections of the Parapharyngeal Space, Arch. Otolaryng. **31**:597 (April) 1940.

he stated that lymphadenitis practically always accompanies acute tonsillitis and peritonsillitis. He assumed that before suppuration occurs in the parapharyngeal space the perivenous lymph glands transmit the infection to the venous channels, which may or may not result in the formation of thrombus. Dixon and Helwig,[9] on the other hand, expressed the belief that the lymphatics play only a minor role, that perivascular abscess formation rarely occurs and that the thrombophlebitis is similar in every way to septic thrombophlebitis in other veins, as in the sigmoid sinus in mastoiditis. It should be recalled that the brain and its coverings are devoid of lymphatics and if this route plays any part in the transmission of infection to the brain it must occur only in the immediate vicinity of the primary site of infection. Actual invasion of the cerebral tissues must take place by another avenue.

1. The perineural lymph channels open directly into the subarachnoid spaces endocranially, and the perineural sheaths may therefore act as avenues through which infectious organisms may gain entrance to the subarachnoid system. A recent study by Kramer and Som[10] indicates that such an eventuality is exceedingly rare.

2. As far as the tissue spaces are concerned, the superior constrictor muscle and its fascial cover separate the tonsillar fossa from the parapharyngeal space — a potential space of great surgical significance. The styloid process separates the space into a smaller, prestyloid, and a larger, poststyloid, compartment. The anterior space, the pharyngomaxillary fossa, is in immediate contact with the tonsillar fossa. The poststyloid compartment contains the great cervical vessels and nerves. When the infection has penetrated into the parapharyngeal space, cellulitis may spread by direct extension toward the base of the skull and the infection enter the cranium through (*a*) a congenital dehiscence, (*b*) an inflammatory erosion in the bony wall or (*c*) venules in the haversian canals in the cranial bones.

Wessely[11] showed that 15 cc. of india ink injected in the parapharyngeal tissue in every instance reached the base of the skull and then extended backward to the foramen ovale, infiltrating both the internal and the external pterygoid muscle and the tissue around the eustachian tubes. He stated that there are three possible routes of infection from the tonsil to the middle cranial fossa: (1) the loose connective tissue of the pharyngomaxillary space, (2) the venous net and (3) a combination

9. Dixon, O., and Helwig, F. C.: Thrombophlebitis of the Internal Jugular Vein as a Complication of Tonsillitis: Study of Six Cases, Ann. Otol., Rhin. & Laryng. **39**:1137 (Dec.) 1930.

10. Kramer, R., and Som, M. L.: Intracranial Pathways of Infection from Diseases of the Sphenoid and Ethmoid Sinuses, Arch. Otolaryng. **32**:744 (Oct.) 1940.

11. Wessely, E.: Die endokranielle Komplikationen nach Peritonsillitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **9**:439, 1924-1925.

of the two. The end result of such infection, no matter how it starts, is always purulent thrombophlebitis of the cavernous sinus. Under certain conditions osteomyelitis of the bones of the skull appears to be the primary extracranial lesion, the other processes developing subsequent to the bony lesion.

In a case of peritonsillar abscess reported by Wylie and Wingrave [12] autopsy revealed extensive necrosis of the parapharyngeal structures on the right side with subsequent extension to the prevertebral muscles, resulting in extensive osteomyelitis of the basioccipital, basisphenoid and ethmoid bones. There was extensive meningoencephalitis in association with bilateral thrombosis of the cavernous sinus. The fact that the right internal jugular vein, jugular bulb and lateral sinus were thrombosed without involvement of either the superior or the inferior petrosal sinus makes one assume that the intracranial complication followed as a direct extension from the base of the skull.

3. The arterial channels supplying the skull and its contents are the internal carotid and vertebral arteries. In cases of generalized pyemia the organism enters the arterial stream and is conveyed by it to all parts of the body and may thereby cause meningitis, multiple abscesses of the brain or thrombosis, depending on the site of lodgment within the cranium.[13]

In cases of chronic endocarditis, for example, the organisms that have been in a dormant state may, under provocation of an acute infection of the upper respiratory tract or after operation in the throat, be reactivated, causing generalized pyemia. The tonsillar region is endowed with a rich blood supply, both the external and the internal carotid artery being in close proximity to the tonsillar fossa. The infection may follow the perivascular structure, or it may gain entrance directly into the arterial stream.

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12. Wylie, A., and Wingrave, W.: Peritonsillar Abscess Followed by Osteomyelitis, Necrosing Encephalitis and Meningitis, Lancet **1**:178 (Feb. 1) 1919.

13. Illustrative of the possible indirect involvement of the cranial contents after tonsillar infections is the case reported by I. H. Tumpeer and A. Levinson (Fatal Infections Following Tonsillectomy, J. A. M. A. **80**:20 [Jan. 6] 1923), in which a boy of 7 presented symptoms suggestive of chorea two weeks before tonsillectomy. On the second postoperative day his temperature rose to 105 F. and he became delirious. The temperature was septic in type for two weeks. He then presented signs of meningitis, and cultures both of the blood and of the spinal fluid yielded a type I pneumococcus. The patient died the following day. At autopsy, both ventricles of the heart were observed to be increased in size. The aortic valve was ulcerated. The lower lobe of the left lung was consolidated. The anterior half of the right cerebral hemisphere was covered with a thin layer of opaque, yellow exudate within the subarachnoid space. A small amount of pus was also present in the left middle ear. In this case the septicemic meningitis was evidently of tonsillar origin, but an indirect rather than a direct complication.

Frank and Scheer [14] were able to trace the infection along the periarterial sheath in 2 cases of acute meningitis following severe tonsillitis. These workers showed experimentally that when a certain strain of organism was injected into the sheath of the internal carotid artery at its bifurcation in young dogs the inflammation extended throughout the sheath of the petrous and the cavernous portion. In 1 animal thrombosis of the cavernous sinus was present.

In our second case, which will be discussed in detail, the wall of the left internal carotid artery had undergone pressure necrosis from the abscess in the parapharyngeal space, resulting in fatal hemorrhage. The detached embolus was carried into the middle cerebral artery, ultimately causing encephalomalacia of the region supplied by the occluded vessel. During the operative procedure the wall of the carotid artery may be accidentally injured or inadvertently be punctured by the point of the needle used for the induction of anesthesia and the infecting organism thus be introduced directly into the circulation.[15]

4. Infection occurs most commonly through venous channels in the form of retrograde thrombophlebitis. In the presence of an acute bacterial invasion, as was pointed out by Turner and Reynolds [16] and others, the products of inflammation may actually impair the integrity of the endothelium of the blood vessel, or the vessel may be injured as the result of the operative procedure, as in tonsillectomy or in the process of evacuation of pus from the peritonsillar abscess. In either event the stage is set for the initiation of phlebitis and thrombophlebitis. Fetteroff and Fox [17] showed that thrombosis of the peritonsillar vein, following the operation on the tonsil, is the usual process. Tanaka and Crowe [18] called attention to the fact that the destruction or localized ulceration of the epithelium in a crypt is associated with thrombosis of the blood

14. Frank, I., and Scheer, C.: Sheath of the Internal Carotid Artery: A Route for Infection from Primary Lesion, Ann. Otol., Rhin. & Laryng. **46**:913 (Dec.) 1937.

15. An example of intracranial complication of needle puncture of the wall of the carotid artery incident to induction of local anesthesia is the case of W. W. Cadbury and A. C. Siddal (Cerebral Abscess Following Tonsillectomy, China M. J. **44**:910 [Sept.] 1930). A tonsillectomy was done on a 41 year old missionary with the use of local anesthesia. On the fifth postoperative day the patient was suddenly seized with jacksonian convulsions. Autopsy disclosed multiple abscesses in the right frontal, parietal and temporal lobes. It was concluded from the distribution of the abscesses that septic material had been carried into the carotid blood current by the anesthetic needle.

16. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Disease, London, Oliver & Boyd, 1931, pp. 15-16.

17. Fetteroff, G., and Fox, H.: The Reaction of Paratonsillar Tissue to Tonsillectomy: Study of Etiology of Post-Tonsillectomy Pulmonary Abscess, Am. J. M. Sc. **166**:802 (Dec.) 1923.

18. Tanaka, M., and Crowe, S. J.: Direct Blood Stream Infection Through the Tonsils, Arch. Int. Med. **33**:473 (April) 1924; Arch. Otolaryng. **1**:510 (May) 1925.

vessels that are normally present in this epithelium. It is easy to imagine, though difficult to prove, that some emboli containing bacteria may enter the blood stream direct, in addition to those which gain entrance through the large lymphatic channels in the tonsils.

It should be remembered that thrombosis is nature's protective mechanism, but in some unfortunate cases the thrombophlebitic process may, failing as a barrier, actually be the means of conducting the infection to the cranial space. The course of the disease and its complications are largely influenced by (1) the anatomic structures through which the infection is carried, (2) the kind of organism and its virulence, (3) the individual resistance and (4) the treatment instituted.[19]

In cases of venous thrombosis following tonsillitis there are two principal paths by which the infection travels to the cranial chamber: 1. By the mesial route, the thrombus in the tonsillar vein may extend into the pterygoid plexus, from which it reaches the cavernous sinus by way of (*a*) the veins of Vesalius, of the foramen ovale and the foramen lacerum, (*b*) the sphenoid bone, or (*c*) one of the communicating branches of the ophthalmic veins.[20]

2. By the lateral route, the thrombus may extend to the internal jugular vein, which may or may not be involved in the thrombophlebitic process, from whence infection may be carried either (*a*) directly to the intracranial blood spaces through the lateral sinus or (*b*) toward the torcular and thence to other portions of the cerebral sinuses.[21]

REPORT OF CASES

CASE 1.—*Cervical adenitis complicating acute tonsillitis in a Japanese man. Repeated surgical evacuation of pus from a cervical abscess. Development of*

19. C. Hall (Sepsis Following Pharyngeal Infections, Ann. Otol., Rhin. & Laryng. **48**:905 [Dec.] 1939) called attention to the fact that the prognosis is better with Streptococcus haemolyticus, and possibly the pneumococcus, than with the staphylococcus or with Streptococcus viridans. The last two organisms are thought to have a greater tendency to metastasize.

20. Both W. P. Eagleton (A New Classification of the Bones Forming the Skull, Arch. Otolaryng. **24**:158 [Aug.] 1936) and H. P. Mosher (Talk to House Officers and the Pharyngomaxillary Approach to the Tip of the Temporal Bone, Ann. Otol., Rhin. & Laryng. **49**:3 [March] 1940) stated that infection of the marrow space of the sphenoid bone is transmitted to the adjoining petrous apex even more readily than from the pneumatized portion of the mastoid process. According to Mosher, osteomyelitis of the body of the sphenoid bone is more common than is generally considered. The dura over the sphenoid is frequently involved secondary to non-fulminating infection from the mucous lining of the sphenoid air cavity.

21. On the basis of his personal experience, Brunner[8] contended that the thrombophlebitic process does not usually extend from the smaller veins within the peritonsillar tissue and the parapharyngeal space into the larger internal jugular vein, there to establish thromboplebitis. In 8 to 9 per cent of a series of 85 cases of deep infection of the neck, presented by Waldapfel, Beck and himself, evidences of thrombophlebitis of the internal jugular vein were discovered.

abscess of the scalp. Evidences of increasing intracranial pressure. Jacksonian seizures on right side. Death. Thrombosis of the superior longitudinal sinus; abscess of the left parietal lobe.

History.—A Japanese man aged 43 consulted one of us (Dr. Hara) on March 13, 1936, for a swelling under his right ear of about two weeks' duration. He gave a history of otitis media on the right side in August 1935, which cleared up in a few days. This was followed in September by right-sided neuralgic headache, which also disappeared in a few days. In December acute rhinitis developed. This was associated with what his family considered to be acute tonsillitis. The infection of the upper respiratory tract cleared up in the early part of January 1936, although his left nasal passage continued to be more or less obstructed and at times there was a profuse nasal discharge. He was fairly well until February 27, when he noticed a swelling in the right side of the neck. Instead of seeking proper medical aid he fell into the hands of a quack. Because the condition became progressively worse the patient finally came to one of us for advice and treatment. He was immediately admitted to the White Memorial Hospital with a diagnosis of cervical abscess on the right side secondary to tonsillar infection.

His past history was irrelevant.

Physical Examination.—The patient was poorly nourished and only moderately well developed. His mind was clear, and he was cooperative during the examination. He weighed 125 pounds (56.8 Kg.) and was 5 feet 5 inches (165 cm.) in height. The temperature on admission was 100.8 F., the pulse rate 120 and the respiratory rate 20 per minute. The veins on the sides of the forehead were much dilated. There was no palpable mass or tender area in the head. The nose was externally normal. The right nasal passage was clear, but the mucosa on the left side was moderately injected and a quantity of mucopurulent discharge was noted on the floor. The movements of the eyes were normal. The pupils were dilated but they reacted to light and in accommodation. He complained of distortion of objects on account of the blurring of vision of the left eye. There was nothing remarkable about his ears. Several teeth were missing, and those remaining were loose and presented definite evidence of pyorrhea. The tongue was coated, but there was no impairment of movement. The left faucial tonsil was of moderate size and was submerged beneath the reddened anterior pillar. The right tonsil was large. Immediately behind its posterior pillar was a small ulcerated area, 12 by 10 mm. in diameter, from which thin pus was draining. The rest of the pharyngeal wall was in a state of diffuse congestion. The vocal cords were salmon colored; they moved freely and met in the median line. The laryngeal mucosa showed a moderate amount of congestion. The thyroid gland was not enlarged.

The left side of the neck presented nothing abnormal, but the right side was brawny and indurated, the subangular space being obliterated. Although no fluctuating mass was felt, one could scarcely mistake the presence of a deep cervical abscess, the origin of infection being interpreted as in the tonsillar region.

There was nothing remarkable about the thorax or abdomen.

A blood count disclosed 3,000,000 red cells and 27,500 white cells per cubic millimeter, of which 8 per cent were lymphocytes and 92 per cent were polymorphonuclears. The hemoglobin concentration by the Sahli method was 66 per cent. The Wassermann reaction of the blood was negative.

Operation and Course of Illness.—On March 14, with the patient under gas and ether anesthesia, about 8 cc. of thick, creamy pus was evacuated, the incision extending from the tip of the right mastoid process obliquely downward along the anterior border of the sternocleidomastoid muscle to the middle third of the

neck. Culture showed pneumococcus type III. After the operation and until March 19 the temperature remained almost normal and the pulse rate varied between 80 and 100 per minute. He slept and ate well. He was permitted bathroom privileges and appeared greatly improved.

On March 20 his temperature rose to 100 F., and the next day a swelling appeared over the retroauricular region. The temperature that evening was 101.6 F.

On March 22 a second operation was performed, with the patient under general anesthesia. An incision was made ½ inch (1.27 cm.) below and slightly behind the right mastoid process. The abscess cavity apparently communicated beneath the sternocleidomastoid muscle with the one which was evacuated at the time of the first operation.

On March 23 the blood count showed 3,160,000 erythrocytes per cubic millimeter, with the hemoglobin concentration 63 per cent by Sahli's method. There were 29,800 leukocytes per cubic millimeter, with a differential count of 12 per cent lymphocytes, 1 per cent monocytes and 87 per cent polymorphonuclears.

On March 27, 500 cc. of whole blood was given by the multiple syringe method.

From March 27 to April 1 the temperature continued to be septic and fluctuated between 97.2 and 102 F. The patient complained of headache.

On April 2 a fluctuating mass, about the size of a silver dollar, could be felt in the vertex of the skull. Two longitudinal incisions, parallel to the parietal suture, were made. A quantity of pus was evacuated from this area. Culture showed pneumococcus type III.

On April 7 a fourth operation was performed for drainage of pus along the posterior border of the sternocleidomastoid muscle in the midcervical portion. All this time the blood cultures had been consistently sterile.

On April 21 the scalp wound continued to discharge a small amount of seropurulent exudate. The fistulous tract in the neck threatened to close. This was reopened and drainage reestablished.

From April 24 to May 10 the patient was up and about, sedatives being given for control of insomnia. He complained of blurring of vision. He felt tired at times and had persistent aching pains over the brows and forehead.

On May 10 Dr. Leland R. House, of the department of ophthalmology, made the diagnosis of acute papillitis.

On May 11 Dr. Herbert G. Childs Jr., of the department of dental surgery, recommended the removal of the remaining teeth. This was done on May 13, with local anesthesia.

On May 18 the visual acuity and visual fields were rechecked. Cellulitis of the scalp persisted, and the fistulous tract in the neck continued to discharge a small amount of pus each day.

On May 20 spinal puncture was made, with the patient lying on the right side. The initial pressure was over 420 mm. of fluid. The technician was unable to make the Queckenstedt test. The spinal fluid was clear. The total cell count was 136 per cubic millimeter. Globulin was slightly increased. There were 80 per cent polymorphonuclears and 20 per cent small lymphocytes. No bacteria were encountered. A culture showed no growth in three days.

On May 26, early in the day, the patient complained of tremor in the right hand. In the afternoon, rather suddenly, he had a jacksonian convulsion affecting the right hand, which was followed by monoplegia brachiofacialis and motor aphasia.

On May 28 the patient gradually regained consciousness. The right arm continued to be flaccid. Speech remained defective. The erythrocytes numbered

3,940,000 and the leukocytes 16,700 per cubic millimeter, of which 12 per cent were lymphocytes, 82 per cent polymorphonuclears and 6 per cent eosinophils. The hemoglobin reading was 80 per cent by Sahli's method.

On May 29 a second spinal puncture was performed to determine whether there was any blood in the spinal fluid. Dr. Thomas Judefind, of the department of pathology, reported that the spinal fluid was clear and contained 9 lymphocytes per cubic millimeter. There was no globulin. The initial pressure was 350 mm.

On May 31 the sensorium was dull. The reflexes were flaccid.

On June 1 the patient became stuporous, the pupils were equal and reacted to light and papillitis persisted. There was marked weakness of the right arm. A blood study showed the leukocytes to be 17,800 per cubic millimeter, of which 10 per cent were lymphocytes, 89 per cent polymorphonuclears and 1 per cent eosinophils. The deep reflexes were sluggish; a definite Hoffmann sign was elicited. There were no pathologic toe signs and no slowing of the pulse rate. Roentgenograms of the skull indicated a fine mottling in the vertex, suggestive of early osteomyelitis.

On June 3 the patient was less stuporous, although paralysis of the right arm persisted. The deep reflexes were sluggish throughout. There was considerable swelling of both eyelids, suggesting interference with the intracranial drainage of the venous system.

On June 4 the leukocyte count had decreased to 8,600 per cubic millimeter, of which 6 per cent were lymphocytes, 94 per cent polymorphonuclears and 11 per cent juvenile and stab forms. The patient had a slight convulsive seizure in the afternoon. He failed to gain consciousness and died at 6: 40 p. m., more than three months after the onset of the tonsillitis.

Autopsy.—The study of the brain was performed by one of us (Dr. Courville). On reflecting the scalp in the usual manner, an area over the vertex of the skull surrounding the parieto-occipital and the sagittal suture proved to be the seat of an infectious process characterized by the formation of granulation tissue, extending down from the scalp to the bone. Small pockets of seropurulent material in the subaponeurotic tissue were opened. The bone in this region presented a slightly yellowish color. Blood exuded from the surface, suggesting an early stage of osteomyelitis of the skull. The pericranium was densely adherent to the skull in this region. The parietal emissary vein passed into the skull by way of a fairly large opening just to the right of the sagittal suture. The interior table of the skull presented only a slightly yellowish appearance in the area corresponding to that described externally.

There was considerable yellowish green discoloration over the right middle ear, giving the appearance of some inflammatory process. The petrous portion of the temporal bone was removed, disclosing a serous exudate in the middle ear and in the adjacent cells, from which smears were taken. There was no evidence, however, of direct extension into the intracranial space, nor was there any dural lesion that could be made out in this vicinity. The petrous portion of the left temporal bone was also removed, but the middle ear here was only slightly moist and the adjacent mastoid cells were entirely dry.

The dural sinuses were completely occluded with an organized blood clot, the organization having proceeded to a point at which the material had a fleshy appearance and texture. This process seemed to be of longest standing in the superior longitudinal sinus, where this channel was completely filled with an organized thrombus, which extended well forward toward the frontal poles. The process also extended downward into the right lateral sinus, well down toward the jugular bulb, where a more recent type of clot was evident, one which had not yet undergone

organization. Examination of the jugular vein of the neck through the still open incision disclosed this vessel to be entirely free of any clot, indicating that the trouble began above in the superior longitudinal sinus, as had already been suggested by the age of the thrombus in that location. An organized thrombus was also found in the left lateral sinus, extending down to the jugular bulb and into the straight sinus, where it entered the great vein of Galen. In these locations the organized thrombus presented the same gross characteristics as those of the thrombus in the superior longitudinal sinus. The superior and inferior petrosal sinuses were free of thrombus.

The dura mater over an area of approximately 5 cm. in diameter was adherent to the cortex of the brain in the left superior parietal region, adjacent to the superior longitudinal sinus. This adhesion was firm. In the leptomeninges surrounding it was what appeared to be a purulent exudate. The underlying brain was fluctuant, as though a relatively large, recent abscess was present, although no pus was obtained on preliminary puncture.

On reflection, the dura in this area was found to be intimately adherent to the brain. On its inner surface was an irregular patch of subdural exudate. This was so firmly adherent to the leptomeninges that it could be peeled off only with some force. A layer of this tissue was also adherent to the leptomeninges. This exudate was almost completely organized and could be removed from the brain only by lacerating it. This area of subdural infection measured 7 cm. in its greatest anteroposterior extent and about the same in its greatest vertical extent.

The convolutions of the brain were markedly flattened, particularly on the left side and over the left postfrontal and parietal areas. The vessels seemed to have a faint amount of streaking along their course. The leptomeninges in the superior parietal lobule likewise were occupied by exudate.

The brain was cut in a series of horizontal sections; the first, passing about 2 cm. below the vertex of the brain, showed diffuse softening of the centrum of the left parietal lobe with cyst formation. In this softened area were two small, circumscribed and fairly heavily encapsulated abscesses (fig. 1). One measured 1.6 by 0.7 cm. and the other 1.3 by 4 cm. The cyst in the outer part of the lobe, filled with coagulated fluid, measured 1.6 by 0.9 cm. in its greatest horizontal diameter. The next section, passing through the corpus callosum, showed diffuse swelling of the entire centrum of the parietal lobe. A number of petechial hemorrhages were present in this lobe. The right hemisphere appeared to be entirely free of any lesion. The ventricular pattern was displaced to the right, incident to the edema and inflammation of the left parietal centrum.

The apparent route of invasion of the brain is shown in figure 2.

One wonders, in retrospect, what the attending surgeon might have done to prevent the development of such a tragedy from what appeared to be originally a trivial infection in the tonsillar region. There can be no doubt that the previous attack of acute otitis media, long-continued oral sepsis and an acute exacerbation of chronic tonsillitis, associated with acute rhinitis and complicated with suppurative sinusitis, must have drained a great deal of the patient's vitality and left him in a state of debility, with little or no resistance against the invading organism. His delay in seeking proper medical care and the failure of early evacuation of the deep cervical abscess were also contributory factors.

The presence of the type III pneumococcus, with its extreme virulence and tendency to produce metastasis and intracranial complications, was indeed a deciding factor in the chain of unhappy events which preceded the days of sulfapyridine and specific serum therapy. That the primary infection arose in the right tonsillar fossa appears to be certain, since for a number of days a small ulcerated area just posterior to the right palatopharyngeal muscle continued to emit a mucopurulent discharge. There are several interesting features in this case concerned with the path of retrograde infection. First, it is noteworthy that the jugular vein remained free from thrombus, although earlier in the course of

Fig. 1 (case 1).—Horizontal section of the left cerebral hemisphere, showing small abscesses in an area of encephalitis in the left parietal lobe.

the illness the infection came in immediate contact with it, while the smaller channels, first a branch of the occipital vein and then the parietal emissary vein in the vertex of the skull, became thrombosed. Later this paved the way for low grade osteomyelitis, which in turn ended in the subcortical abscess of the parietal lobe.

Second, as far as we can learn from the review of literature, this is the only instance recorded in which an ascending infection from the cervical region passed into the cranium through a relatively small emissary vein to involve the hemisphere on the side opposite that in which the infection began. It is difficult to explain why the infection did not proceed from the right internal jugular vein into either the

inferior or the superior petrosal sinus and then invade the cavernous sinus, as most authors have reported. It is equally difficult to explain, since the primary infection occurred in the peritonsillar structures, why the thrombosis did not progress in a more direct course by way of the pterygoid plexus and, finally, into the cavernous sinus. Dixon warned against undue trauma to the soft tissues of the neck and throat in surgical manipulation in cases of peritonsillar abscess. With this opinion we are in thorough accord. Sharp cutting instruments were used instead of blunt ones. Every effort was made to avoid tearing the structures

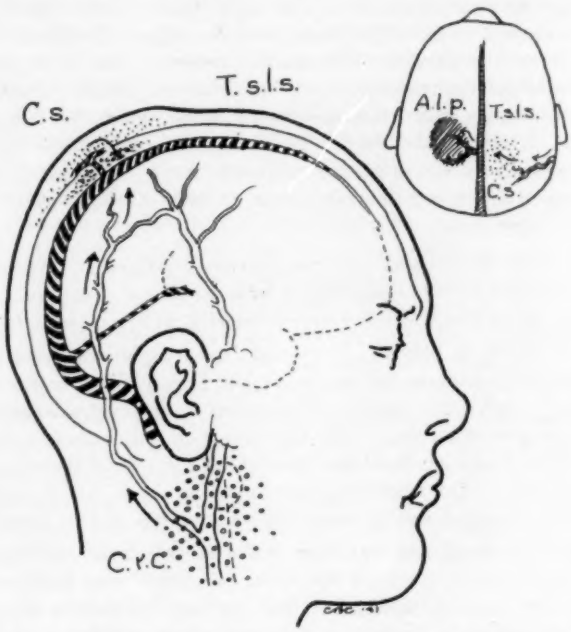

Fig. 2 (case 1).—Schematic drawing, showing probable course of extension from the tonsil to the brain. A peritonsillar abscess set up adjacent cellulitis of the upper cervical region (*C.r.c.*), from which extension to the scalp took place, probably by vein. Local cellulitis of the scalp (*C.s.*) gave rise to regional osteomyelitis and thrombosis of the parietal emissary vein. Thrombosis of the superior longitudinal sinus (*T.s.l.s.*), with secondary involvement of the superior cerebral veins, resulted in formation of abscess in the parietal lobe (insert, *A.l.p.*).

about the abscess. In opening the abscess on the vertex, two parallel incisions were made in the axis of the superior longitudinal sinus about ½ inch away from the center in order to avoid the suture line.[22]

22. We are familiar with Mosher's technic in the management of acute osteomyelitis of the skull. Our judgment made us institute conservative measures rather than sacrifice a large portion of the parietal bone, with the attending risk of shock

(Footnote continued on next page)

Banti [23] reported a similar case in which occlusion and rupture of the meningeal veins led to infective cerebral phlebitis and encephalomalacia after purulent tonsillitis.

His patient was a girl 17 years of age, who three weeks before had been admitted to the hospital, suffering with intense right-sided headache and myalgia. In a week her headache became progressively more severe and was accompanied by a series of jacksonian convulsions. At the time of her admission there were slight hemiparesis and dysarthria but no ocular disturbances. Her mind was clear. There was no evidence of meningitis, as shown by the study of the spinal fluid. At autopsy the cavernous sinuses were observed to be completely free from thrombosis. There was no hemorrhage beneath the dura. On the surface of both cerebral hemispheres, the superior cerebral veins, which empty into the superior longitudinal sinus, were seen to be largely thrombosed. The pia was reddened, but there was no trace of exudate. Histologic examination of the brain showed diffuse hemorrhagic foci in the left hemisphere, the most pronounced being in the superior parietal lobe and, to a lesser degree, in the frontal lobe. There was no extracranial lesion save the infected left tonsil, which was greatly enlarged. Banti concluded that the purulent tonsillitis was the primary morbid factor in the development of the cerebral thrombosis.

CASE 2.—*Left peritonsillar abscess following influenza. Surgical drainage followed by repeated profuse hemorrhages from the throat. Death in coma. Obstruction of the left anterior and middle cerebral arteries, with cerebral softening.*

History.—On Jan. 2, 1940 an 18 year old Jewish youth was admitted to the Los Angeles County Hospital, in the service of Dr. John Mackenzie Brown, who gave us permission to make this report. The patient complained of trismus and severe pain in the left side of the head. He had been sick with influenza for seven days. Two days before admission there was pain in the left side of the neck, accompanied by chills and fever. On admission his temperature was 104 F. by mouth. His tonsils had been removed and the nasal septum resected several years before.

Examination.—His throat was fiery red and the uvula swollen. There was slight swelling of the left side of the neck, associated with tenderness under the angle of the jaw, but no fluctuation was elicited. A culture of material from the throat disclosed Strep. viridans as the cause of the infection. The Wassermann reaction of the blood was negative.

Course of Illness.—On January 5 a left peritonsillar abscess developed, but no pus was obtained on incision. The temperature subsided. A blood count showed

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and spread of the infection associated with such a drastic procedure, since we did not know the extent of the lesion already present in the intracranial sinuses. Removal of the primary focus of infection probably would have had no direct bearing on the final outcome. Particularly was this true once diffuse pneumococcic cellulitis of the scalp had become established. For this reason no attempt was made to enucleate the tonsils. For the same reason the extraction of the infected teeth, which was done during the course of his illness, probably had no great influence other than to eliminate a focus of infection which the consulting ophthalmologist felt at the time might be the cause of acute papillitis.

23. Banti, G.: Trombosi dei seni della dura madre consecutiva a tonsillite, Clin. med., Pisa **9**:14, 1902; cited by Malan, A.: Tromboflebite del seno cavernoso consecutiva a flemmone peritonsillare, Arch. ital. di otol. **41**:1 (Jan.) 1930.

4,000,000 erythrocytes per cubic millimeter, 80 per cent hemoglobin and 14,000 leukocytes, of which 84 per cent were polymorphonuclears.

On January 11 a profuse hemorrhage occurred from the nasopharynx, the precise site of which could not be determined, although it was evidently not from the peritonsillar incision. His pulse became thready, and a transfusion was given immediately.

On January 12 a second massive hemorrhage, to the extent of almost a liter within five minutes, occurred from the nasopharynx. His blood pressure was 60 systolic and 30 diastolic. A transfusion of 250 cc. of whole blood was given.

On January 13 a third hemorrhage occurred, after which the pulse was weak and rapid. The blood pressure was 90 systolic and 50 diastolic. The bleeding was controlled by a nasal pack, and a third transfusion was given. The patient tolerated the pack poorly. The bleeding point was located in the left side of the nasopharynx.

On January 15 a fourth massive hemorrhage occurred. The postnasal pack was reinserted. The blood pressure was 94 systolic and 50 diastolic. The pulse rate was 104. The hemoglobin content was 40 per cent; the erythrocytes numbered 1,750,000 and the leukocytes 24,000 per cubic millimeter, of which 78 per cent were polymorphonuclears, 14 per cent small lymphocytes, 3 per cent eosinophils and 6 per cent monocytes. A transfusion of 500 cc. of whole blood was given.

On January 16 a transfusion of 500 cc. of blood was again given.

On January 21 a fifth massive hemorrhage occurred, resulting in profound shock. A transfusion of 350 cc. of blood was given.

On January 22 the patient became stuporous. His pulse rate was 96 per minute, and his blood pressure was 110 systolic and 70 diastolic. Babinski's sign was definitely present on the right side and was suggested on the left. There was pronounced bilateral ankle clonus, more pronounced on the right. A blood count disclosed 2,500,000 erythrocytes per cubic millimeter and 12,000 leukocytes, of which 76 per cent were polymorphonuclears. The hemoglobin content was 50 per cent. Another blood transfusion was given.

On January 23 the patient was semicomatose, with a pulse rate varying between 100 and 120 per minute. The blood pressure was 100 systolic and 40 diastolic. The pupils were dilated and the reflexes diminished. Babinski's sign was no longer present.

On January 24 the patient was found to be in profound coma. His temperature rose to 107 F.; the respirations were rapid, and there was pronounced acidosis. There was no rigidity of the neck or spine. The reflexes were hyperactive; later they were diminished or absent. The chest was clear, and there was no dulness.

On January 25 the blood pressure was 14 systolic and 0 diastolic, and the deep reflexes were absent. The patient died that afternoon.

Autopsy revealed a massive blood clot in the left parapharyngeal space and necrosis of the left internal carotid artery, with embolism of the left middle cerebral artery and consequent softening of the left cerebral hemisphere.

The essential lesion of the brain consisted of red softening, indicative of recent arterial occlusion, in the area of distribution of the left middle cerebral artery (fig. 3). This softened area included the frontal portion of the operculum, the precentral gyrus, the posterior portion of the superior and middle temporal gyri, the postcentral gyrus and the adjacent portion of the inferior parietal lobule, the supramarginal gyrus. In the anterior part of this softened area, the cortex was marked with petechial hemorrhages, giving a leaden coloration to the cortex. This area was markedly swollen. The regional veins were obviously thrombosed. There were some small regional thrombi in the superior anastomotic vein and in an inferior cerebral vein, a rather large solitary vein arising from the lateral sinus

on the left side. The entire left temporal lobe was swollen and softened. The middle cerebral artery, which was partly opened, showed a small amount of coagulated material in its first portion, evidently the tail of the thrombus.

Frontal sections of the brain showed swelling of the centrum of both hemispheres, while a section through the anterior part of the temporal lobes revealed

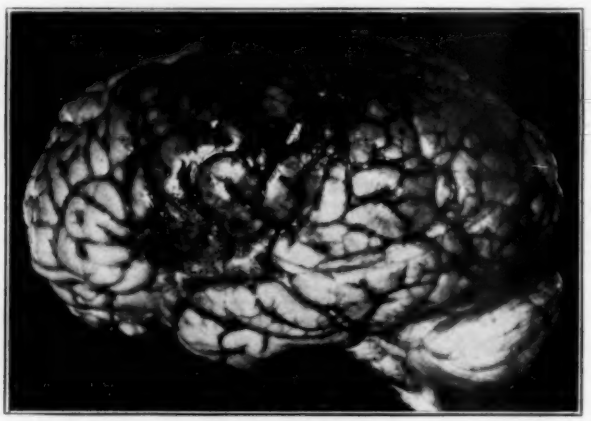

Fig. 3 (case 2).—Softening of the left cerebral cortex consequent to embolic occlusion of the left anterior and middle cerebral arteries.

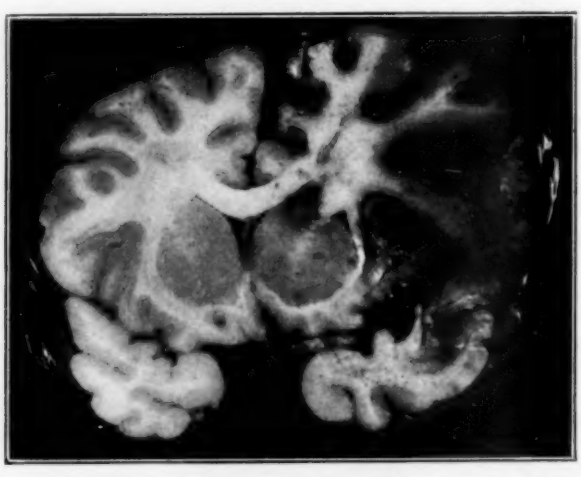

Fig. 4 (case 2).—Coronal section of the brain, showing distribution of red softening. Involvement of the medial aspect of the hemisphere indicates occlusion of the anterior as well as the middle cerebral artery.

characteristic red softening in a fairly early stage (fig. 4). Not all the softening, however, was of this variety. Further sections showed softening of practically the entire superior and lateral portions of the hemisphere, extending into the basal ganglions and the internal capsule and involving the corona radiata and the centrum

of the hemisphere. The softening extended fairly well back into the hemisphere to within the limits of the occipital lobe. Section through the brain stem and cerebellum failed to disclose additional lesions of any sort. Softening of the mesial cortex of the frontal lobe indicated occlusion of the anterior cerebral artery as well.

Routine sections of blocks of tissue from the softened area showed somewhat disorganized cortical and subcortical nerve tissue, which was necrotic and friable and studded with foci of hemorrhage. Areas of necrosis in the cortex, such as are present after asphyxia, were observed. Under higher magnification these areas were seen to be pale and to stain poorly and the cortical blood vessels showed an increased tendency to proliferation, with budding and new vessel formation. What appeared to be foci of infection about some of the blood vessels were much in evidence in the form of collections of polymorphonuclear leukocytes in areas of softening. This would seem to indicate that a suppurative process was superimposed on the softening.

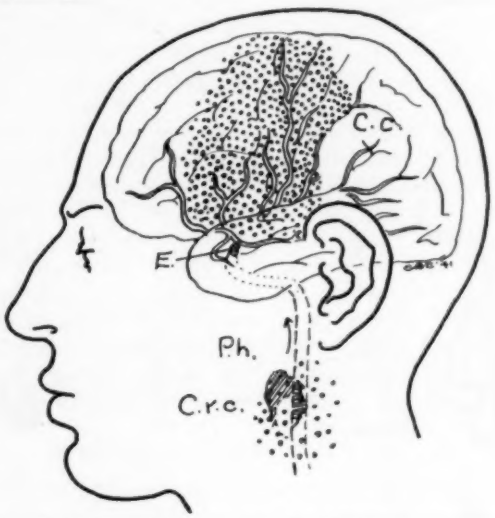

Fig. 5 (case 2).—Schematic drawing, showing probable mechanism of production of cerebral lesion. Peritonsillar infection resulted in local cellulitis (*C.r.c.*) and erosion of the wall of the internal carotid artery, resulting in gross hemorrhage (*P.h.*). Dislodgment of thrombus in the artery resulted in embolic occlusion (*E.*) of the proximal ends of the left anterior and middle cerebral arteries, with consequent cerebral softening (*C.c.*).

The evident mechanism of infection is shown in figure 5.

There are several points of interest in this case which deserve emphasis. First, the primary lesion was apparently an unrecognized abscess in the left parapharyngeal space. Furthermore, the repeated massive hemorrhages did not occur as a result of the incision of the peritonsillar abscess but rather were caused by an ulceration of the internal carotid artery, which in turn was a consequence of thrombophlebitis of the periarterial venous plexus or was a direct result of the arteritis incident to pressure from the abscess or to the effect of local toxins or some unrecognized combination of these factors.

The internal carotid artery, as it passes upward through the parapharyngeal space, is at times in close proximity to the tonsillar bed, forming a so-called false aneurysm. In the presence of such an anatomic anomaly, it is conceivable that the artery might become, under certain circumstances, a most vulnerable structure. Experience has shown that recurrent hemorrhages of increasing proportions, at intervals of from a few hours to several days, invariably presage the oncoming of the fatal episode. After the initial hemorrhage the eroded vessel wall is capable of sealing the source of bleeding. With the return of the blood pressure to the normal level, succeeding hemorrhages of greater magnitude occur, until nature is no longer able to cope with the situation.

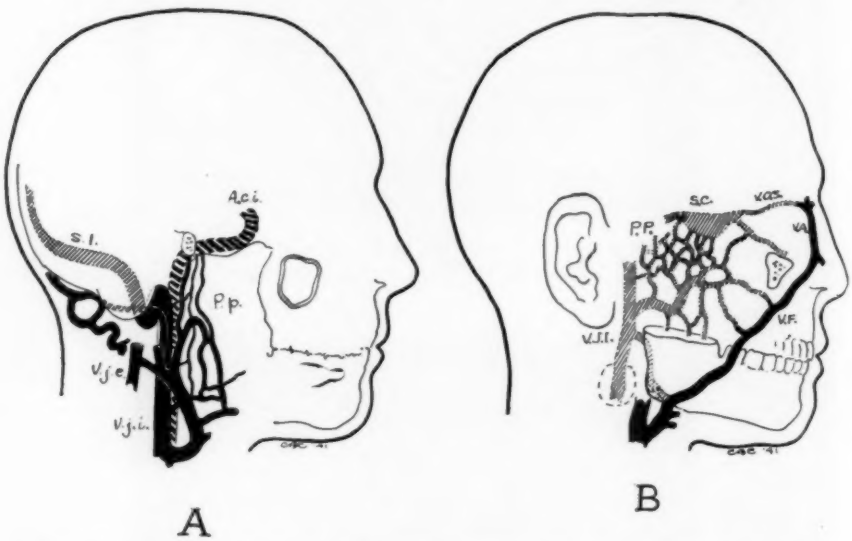

Fig. 6.—Schematic drawings of vascular communications of the tonsillar region with intracranial vessels. *A* shows large vessels immediately in contact with the tonsil and the vascular channels leading to the intracranial space. *V.j.i.* indicates internal jugular vein; *V.j.e.*, external jugular vein; *P.p.*, pharyngeal plexus; *A.c.i.*, internal carotid artery, and *S.C.*, lateral sinus. (For indirect channels, see diagram in figure 2.) *B* shows more indirect communications through the pterygoid plexus and facial vein to the cavernous sinus. *V.j.i.* indicates internal jugular vein; *P.p.*, pterygoid plexus; *V.f.*, facial vein; *V.A.*, angular vein; *V.O.S.*, superior ophthalmic vein, and *S.C.*, cavernous sinus.

When the condition is recognized early, the rational procedure is to ligate the common carotid artery. We are not unmindful of the serious cerebral complications that follow the vascular disturbance, but when the subject is under 40 years of age the chances of permanent trauma are said to be slight. When the source of bleeding is known to be from the external carotid artery, the most ideal procedure is to isolate and ligate the artery. In practice, however, it is not always so simple to identify this vessel

in the bed of inflammatory mass, and one often has to be content to deal with the common carotid artery.

The neurologic manifestations, such as profound shock, altered deep reflexes and coma, must be attributed to acute general and cerebral anemia consequent to the repeated massive hemorrhages. The absence of a thrombophlebitic process in the internal jugular vein and the presence of ulceration, rather than surgical injury, of the carotid artery, independent of the surgical wound, make the case truly unique in medical literature.

The nearest approach to this case was one reported by von Soubiron and Emiliani.[24]

The patient, a man 34 years of age, had undergone tonsillectomy with local anesthesia two weeks before his entrance to the hospital because of hemorrhage. An unrecognized parapharyngeal abscess, secondary to tonsillectomy, had developed. Pressure from the abscess produced ulceration of the external carotid artery. To control the hemorrhage the common carotid artery was ligated. Thrombophlebitis of the internal jugular vein was confirmed at the time of the second operation. On the following day aphasia and hemiparesis appeared. When the collateral circulation was established the neurologic signs disappeared. The second hemorrhage occurred from the same artery through its peripheral extremity after the circulation was reestablished. The bleeding was controlled but the sepsis continued. Three days before death jacksonian convulsions occurred, with a slow pulse rate and reappearance of the aphasia. An incomplete autopsy showed multiple abscess formation in the superior portion of the parietal lobe, with cortical and subcortical localization. The center of the unencapsulated abscess was entirely liquid. Examination of the pus from the abscess showed chains of streptococci, gram-positive diplococci and bacteria. There was associated microscopic meningitis.

SURVEY OF THE LITERATURE

In table 1 the incidence of intracranial complications of tonsillar origin, as discovered in a survey of the literature from 1900 to 1940,

TABLE 1.—*Intracranial Complications of Tonsillar Origin (1900-1940)*

Complication	No. of Cases
Thrombosis of lateral sinus	4
Thrombosis of cavernous sinus	43
Primary septic meningitis	9
Encephalitis, cerebral abscess, etc.	13
Embolism, etc.	7
Total	76

is indicated. A total of 76 definite cases were found. The lesions in these instances included thrombosis of the cerebral venous channels, septic meningitis, encephalitis and brain abscess and, more rarely, vascular accidents, such as cerebral hemorrhage and embolism.

24. von Soubiron, N., and Emiliani, C. M.: Gravísimo y rãro post operatario de amigdalectomía, Rev. Asoc. méd. argent. **52**:721 (July 30) 1938.

THROMBOSIS OF THE LATERAL SINUS

The occurrence of thrombosis of the lateral sinus of otitic origin is so common and the clinical picture is so well established that other possible origins are usually forgotten. There were 4 cases of thrombosis of the lateral sinus of tonsillar origin in this series,[25] in all of which the condition was due to retrograde extension within the internal jugular vein. In the case reported by Banti [23] the inflammatory process involved both lateral and longitudinal sinuses.

In this rare complication of tonsillar infection it seems clear that any direct involvement of the lateral sinus must of necessity follow a retrograde extension of infection through the internal jugular vein; indirectly it may be secondary to cellulitis of tonsillar origin, the lateral sinus being invaded by a thrombus originating in the emissary vein of the mastoid. The lesion itself is no different from that of otitic origin. It may spread extensively in the sinus system and may, in turn, be followed by other secondary intracranial lesions.

THROMBOSIS OF THE CAVERNOUS SINUS

By far the largest number of intracranial complications of tonsillitis proved to be thrombosis of the cavernous sinus. The collected cases are tabulated in this connection (table 2).

TABLE 2.—*Incidence of Thrombosis of Cavernous Sinus of Tonsillar Origin with Associated Complications*

	Thrombosis of Cavernous Sinus	Secondary Meningitis	Encephalitis	Subdural Abscess	Brain Abscess	Recovery
Tonsillectomy and adenoidectomy...	1	0	0	0	0	0
Tonsillectomy	5	4	0	0	0	0
Tonsillotomy	1	0	0	0	0	1
Acute tonsillitis ...	11	7	0	1	0	1
Peritonsillitis	26	20	9	1	2	2
Total	44	31	9	2	2	4

In figure 6 is shown a schematic plan of efferent and afferent vascular communications, both within and without the cranial vault. In addition to those illustrated, there are innumerable unnamed venous plexuses on both the intracranial and the extracranial side of the base of the skull. Because of this freedom of vascular anastomosis and its proximity to the posterior paranasal air sinuses, the cavernous sinus is the most vulnerable site within the cranium for the development of secondary infection.

25. Three patients were females, and the sex of 1 was not stated. One patient was in the second decade of life, 1 in the fifth, 1 in the sixth and the age of 1 was not mentioned. The youngest patient was 18 years of age and the oldest 58.

Thrombosis of the cavernous sinus after tonsillitis may be a direct consequence of the infection per se or may follow operative procedures on the tonsils or adenoids, the operation serving to stir up the infectious process and to open up avenues for venous extension. In either case the route of extension is probably the same. The richness of the venous plexuses of the pharynx and the communications with those of the cavernous sinus explain the more common involvement of this sinus. It is most likely that infection spreads in the pharyngeal plexus of veins to enter the sinus by way of connections in the pharyngomaxillary space or, perhaps less likely, through the inferior ophthalmic vein via the pterygoid plexus. It would be a rare case indeed in which infection followed the more roundabout route of the pericarotid plexus, the facial vein or the internal jugular–inferior petrosal channels.

In the operative cases more direct routes of extension may be opened up, as suggested in the instances to be cited.

CAVERNOUS SINUS THROMBOSIS

Following Operation on Tonsils and Adenoids.—In 7 cases the infection of the cavernous sinus was the primary intracranial complication following operation on either the tonsils or the adenoids or on both. The operative procedures included adenotonsillectomy, 2 cases; tonsillotomy, 1 case, and tonsillectomy, 4 cases. Five patients were males, 1 was a female and the sex of 1 was not stated. One patient each was in the first, the second and the third decade; three were in the fourth, and the age of 1 was not given. The youngest was a boy of 10 and the oldest a woman of 35 years. In 2 cases the operation was performed under general anesthesia and in the other 5 with local anesthesia. In 4 cases the infection appeared to have spread through the pterygoid venous plexus, and in only 1 was the internal jugular vein thrombosed. Secondary meningitis was present in 4 cases. In the case reported by O'Malley (as discussed in Davis'[26] paper) there was osteomyelitis of the body of the sphenoid bone, with direct extension of infection along the optic nerve into the cavernous sinus. The patient in Dean's[27] case was the only one in the series who recovered. Tonsillotomy had been performed with the most meticulous care. Retrograde thrombophlebitis extended from the internal jugular vein through either the inferior or the superior petrosal sinus or through both to the cavernous sinus. The left eye required enucleation, but the boy eventually recovered, after a

26. Davis, H. J.: Chart and Brief Notes of a Case of Cavernous Sinus Thrombosis Following Tonsillitis in a Boy Aged Ten: Fatal Termination in Eighteen Days, Proc. Roy. Soc. Med. **6**:174 (July) 1913.

27. Dean, L. A.: Severe Sepsis Following Tonsil Operation, Laryngoscope **20**:739 (July) 1910.

long illness. It will be of interest to note that when the infection traveled through the efferent venous channels to the cavernous sinus the organism became attenuated and the patient survived.

Three cases of this complication have been recorded by Loeb, but these were omitted from our list because of lack of details in his report. Another case reported by Wales [28] was also excluded because the complications might as well have been secondary to mastoiditis. A 7 year old boy had an adenoidectomy. Immediately on recovery from the ether anesthesia he complained of occipital pain. On the third day tenderness developed over the right mastoid, which was later opened and found to contain pus. The temperature continued to be septic. The right infraorbital region began to swell. In ten days exophthalmos, chemosis of the lids and optic neuritis occurred, which were followed by coma and convulsions. Autopsy revealed bilateral thrombosis of the cavernous sinus, secondary to trauma to the basioccipitalis inflicted by a Beckmann curet in the hands of an inexperienced operator.

Following Acute Tonsillitis.—There were 11 cases in this group. In all but 1 the patients were males. Their ages ranged from 10 to 32 years. One patient was in the first decade of life, 4 were in the second, 3 were in the third and 2 were in the fourth. One was a young man whose age was not given. The patient in Seggel's case [29] (cited by Dean) was the only one of the series who recovered. He was a robust soldier, aged 20 years, in whom cellulitis of the right tonsillar region developed. The right internal jugular vein was thrombosed. He eventually recovered, after an illness of ten weeks, but with total loss of vision. Secondary meningitis was present in 7 cases and subdural abscess in 1. In 3 cases the path of infection appeared to have been through the ophthalmic veins; in 3 the internal jugular vein was thrombosed; in 2 both the pterygoid plexus and the internal jugular vein were involved; in 1 the pterygoid plexus only was affected, and in 1 the pus traveled along the mandibular branch of the fifth cranial nerve and extended through the foramen ovale. In addition to the case recorded here, 2 others were reported by Davis; 1 patient recovered and the other died. On account of lack of sufficient confirmatory evidence, these cases are also omitted from the table. In a case reported by von Kecht [30]

28. Wales, E. de W.: The Occipital Bone in Otolaryngology and Rhinology, Arch. Otol. **37**:1 (Feb.) 1908.

29. Seggel, K.: Erbindung infolge Tonsillitis phlegmonosa auf dem Wege der Thrombosinusitis cerebralis, Klin. Monatsbl. f. Augenh. **14**:129, 1907; cited by Dean.[27]

30. von Kecht, B.: Zur Kenntnis und Behandlung der zum Endokranium aufsteigenden Eiterungen der Mundhöhle und des Rachens, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:275, 1936.

and in another by Lewis [31] the thrombosis was ushered in by otitis and the cases are therefore not included in our list. In Vogel's [32] case there was a coexisting rhinologic lesion, and because of the uncertainty of its origin this case was also excluded.

Following Peritonsillitis and Peritonsillar Abscess.—There were 26 cases in this group. Twenty of the patients were males and 6 were females. The youngest was 2 years old; the other patient in the first decade was 10; the oldest was 61. The largest number were in the second and third decades of life, there being 9 in each. Two were in the fourth decade, 2 in the fifth, 1 in the sixth and 1 in the seventh. Two recovered. The case of 1 of these was reported by Long,[33] in which the thrombophlebitic process spread through the efferent veins of the cavernous sinus. The case of the other was reported by Claus,[34] in which the peritonsillar abscess was incised. At the first appearance of the symptoms of piercing pain in the orbit the eye was enucleated. Secondary meningitis was present in 20 cases, encephalitis in 9 cases, subdural abscess in 1 case and brain abscess in 2 cases. The orbital vein was thrombosed in 3 cases. The internal jugular vein was ligated and excised in 2 cases and ligated only in 3. At autopsy thrombophlebitis was discovered in 6 cases. The pterygoid plexus was thrombosed in 7 cases. Wessely,[35] in his second article, recorded 2 cases which were previously reported and, in addition, mentioned 4 cases, 1 that of a man aged 46, who died of suppurative left peritonsillar abscess, complicated by mediastinitis and ascending infection to the base of the skull. Wessely failed, however, to state the exact nature of the endocranial lesion.

In the case reported by Calamida [36] the condition was associated with dental extraction, after which peritonsillitis developed and incision was required. Still another case recorded by Loeb lacks verifying

31. Lewis, R., Jr.: A Case of Cavernous Sinus Thrombosis Following an Acute Tonsillitis: Death; Report of Autopsy Findings, Ann. Otol., Rhin. & Laryng. **18**:921, 1909.

32. Vogel, K.: Aufsteigende Infekten des Endocraniums bei tonsillogener Rachenphlegmone, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:4, 1933.

33. Long, J. W.: Excision of the Internal Jugular Vein for Streptococcic Thrombi of Vein and Cavernous Sinus Causing Paralysis of Orbital Muscles, Surg., Gynec. & Obst. **14**:88 (Jan.) 1912.

34. Claus: Exentratio Orbitae bei drohender Thrombose des Sinus cavernosus infolge tonsillogener und dentaler Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **36**: 315, 1934.

35. Wessely, E.: Ein weiterer Beitrag zur Entstehung der endokraniellen Komplikation nach Tonsillitis und Peritonsillitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:167, 1931.

36. Calamida, U.: Trombosi del seno cavernoso e del seno longitudinale superiore consecutivo ad ascesso peritonsillare, Osp. maggiore **18**:57 (Feb.) 1930.

evidence, and these are all excluded from our series. Malan [37] reported a case of thrombosis of the cavernous sinus of tonsillar origin and stated that he had been able to collect only 23 cases. Among them were those of Ogle, Mosher and Mayer. A diligent search has thus far failed to locate the reports of these authors.

Complicated by Encephalitis and Brain Abscess.—There were 12 cases in this series. Ten of the patients were males and 2 females. In 1 case the lesion followed tonsillitis, and in 9 cases peritonsillitis or peritonsillar abscess was the source of infection. Purulent meningitis was reported to be present in all cases, encephalitis in 9 cases, subdural abscess in 1 and brain abscess in 2. It is worthy of note that thrombosis of the cavernous sinus does not usually result in abscess of the brain. In the case of Wylie and Wingrave the abscess occurred from extension of the osteomyelitis of the basioccipital and basisphenoidal bones secondary to extensive necrosis within the pharyngomaxillary space. The internal jugular vein was thrombosed in 6 cases, in 1 of which the vein was ligated. The pterygoid plexus was involved in 5 cases, and in 1 case, which was reported by Rössle,[38] both the internal jugular vein and the pterygoid plexus were thrombosed. Von Kecht reported a case of abscess of the temporal lobe as a complication of peritonsillar abscess. The lad was ill only eleven days, and since this period is too short for an abscess to develop, we are inclined to believe the lesion was encephalomalacia. His second case was that of a girl aged 6 years who had intracranial complications secondary to acute otitis following tonsillitis. (At best this is an example of an indirect complication of tonsillitis.) In Witchell's [39] case an extensive superficial abscess of the temporoparietofrontal region and of the cortex of the cerebellum was reported. The first lesion was apparently a subdural abscess. There was no evidence to prove that the subarachnoid space was infected. In the case reported by Kernan [40] the abscess of the left temporosphenoid region was most likely secondary to thrombosis of the lateral sinus, rather than the direct result of tonsillar origin.

It is extremely interesting to note that the highest incidence of thrombosis of the cavernous sinus occurred after peritonsillitis, less often after acute tonsillitis and still less often after operations on the tonsils. Secondary meningitis, encephalitis and brain abscess occurred in the order of incidence named. In view of such observations one can

37. Malan, A.: Tromboflebite del seno cavernoso consecutiva a flemmone peritonsillare, Arch. ital. di otol. **41**:1 (Jan.) 1930.

38. Rössle, R.: Pathologisch-anatomische Demonstrationen aus dem Gebiete der Halskrankheiten, Internat. Zentralbl. f. Ohrenh. **25**:223, 1926.

39. Witchell, I. S.: Abscess of the Brain Following Tonsillitis and Retropharyngeal Abscess, Arch. Otolaryng. **29**:835 (May) 1939.

40. Kernan, J. D.: Cavernous Sinus Thrombosis, Secondary to Peritonsillar Abscess, Laryngoscope **31**:259 (April) 1921.

reasonably assume that in cases of peritonsillitis and peritonsillar abscess there exists (1) greater virulence of the organism, (2) lack of local tissue immunity, (3) greater tendency for the organism to spread, (4) failure to limit the infection within the sinuses and (5) earlier involvement of the pial veins, from which infection spreads to the meningeal vessels.

In this series there were 4 cases of recovery. In all but 1 case the infection was of the chronic type. In cases reported by Dean, Seggel and Long the infectious process extended from the internal jugular vein and upward and thence through either the inferior or the superior petrosal sinus to the cavernous sinus. Owing either to the avirulence of the organism or to the unusual resistance of the host, during their sojourn in the blood stream the bacteria became attenuated and the patients survived, the interval of convalescence varying up to more than two months. But all of them lost their eyesight, either totally or in part. In the fourth case, reported by Davis, the patient recovered. No mention was made of the path of infection or the chronicity of the disease.

In only 1 case, that of Claus, did the patient recover after an acute infection, and then only after the affected eye was sacrificed. Claus stated that in his study of 160 cases of thrombosis of the cavernous sinus originating from either the tonsils or the teeth, the infection spread by way of the orbit in 5, and in all of these it ended in death. In each case the patient first complained of sudden piercing pains in the eye on the affected side, with marked increase of the pain when pressure was exerted on the closed eye. The next symptom was edema of the conjunctiva, followed by protrusion of the bulb and in most cases by the same sequence of events on the other side. The outcome was always fatal. Claus now enucleates the eye just as soon as the piercing pains occur in the eye, even though the ophthalmologist may fail to find anything unusual.

ACUTE PRIMARY MENINGITIS

In certain cases of acute tonsillitis, peritonsillitis and postoperative involvement in this region, the organisms or their toxins or both gain access to the meninges, as in otogenic and rhinogenic infections. The lesion produced may be toxic, thrombotic or infectious. The final outcome is a transitory serous or reactive meningitis or suppurative meningitis, depending on the degree and extent of invasion. Kroll [41] is the only one to speak of tonsillitis as a possible cause of acute serous meningitis.

Meningitis of one type or another is a common disease of childhood. To associate one common disease with another common disease or with

41. Kroll, M.: Die neuropathologischen Syndrome, Berlin, Julius Springer, 1929, p. 456.

an operation, which is also common, does not imply that they are related as cause and effect. In the presence of overwhelming sepsis, particularly when the resistance is low, meningitis is a common outcome. In cases of infection of the blood stream the meninges may be the seat of a primary intracranial lesion. In a large number of instances, however, meningitis is usually secondary to thrombophlebitis of the cerebral sinuses or extension from osteomyelitis or is associated with abscess of the brain. The infection travels through preformed bony canals or follows the blood vessels or nerve sheaths. In a few instances, as will be shown, the pus gains entrance to the cranium by direct destruction of the bone.[42] If, under these circumstances, the infection is of low virulence and the resistance of the host is sufficiently high, a protective thickening of the dura is produced and the organism is thus excluded, although an extradural abscess may be produced. In acute fulminating leptomeningitis so overwhelming is the invasion that the brain is soon covered with

TABLE 3.—*Incidence of Acute Primary Meningitis of Tonsillar Origin*

	Patients with Primary Meningitis	Patients Recovered	Males	Females	Sex Unknown
Adenoidectomy	1	0	1	0	0
Tonsillectomy	3	1	1	1	1
Tonsillitis	2	0	0	2	0
Peritonsilitis	3	0	1	2	0
Total	9	1	3	5	1

purulent exudate. The meningeal vessels are greatly dilated. The bulk of the exudate is found at the base, where it fills the cisternal spaces.

Table 3 shows the incidence of meningitis as a consequence of tonsillar infection. There were 9 cases of verified primary meningitis, in 3 of which the patients were males, in 5 females and in 1 the sex was not given. One patient was in the first decade of life, 4 were in the second and 3 in the third, and the age of 1 was not stated. The youngest patient was a 7 year old girl and the oldest were 2 men, each 30 years of age. In 1 case meningitis followed adenoidectomy, in 1 osteomyelitis of the base of the skull from direct extension of a retropharyngeal abscess in the course of acute exacerbation of chronic tonsillitis, in 1 acute tonsillitis, in 3 tonsillectomy with local anesthesia and in 3 peritonsillitis. Because of lack of verification by bacteriologic studies, we have purposely

42. In a paper presented before the annual session of the American Laryngological, Rhinological and Otological Society, June 17, 1941, entitled "The Newer Conceptions of Rhino-Otological Meningitis," James G. Dwyer, on the basis of an analysis of 617 verified cases of meningitis, stated that osteomyelitis of the skull is one of the chief factors in the causation of meningitis.

omitted the cases reported by Loeb (2 cases); Thurley [43]; Putnam (2 cases) [44]; Leatherwood; Keen (2 cases); Nesbitt, Tumpeer and Levinson; Dean; Hahn [45]; Jennemann (3 cases) [46]; Siemerling [47]; Fränkel,[48] and Wessely [35] (fourth case). In the case reported by Smith and Scott [49] the disease was otitic in origin. In Jacobsen's [50] case meningitis was secondary to bacterial endocarditis, which developed six weeks after tonsillectomy. In Proskauer's case [51] the meningitis is said to have developed after dental infection coincident with peritonsillitis. These cases are also omitted, for obvious reasons. Only 1 patient recovered in this series, and her case was reported by Harris and Yenikomshian.[52] The spinal fluid was turbid, with a cell count of 540 per cubic millimeter; culture disclosed a pneumococcus (not type III), and specific serum was given. In addition, there were 7 cases of aseptic meningitis in which recovery occurred; 1 each was reported by Lewin [53] and Neumann,[54] 2 by Reisman [55] and 3 by Frühwald,[56] in all of which the meningitis was secondary to tonsillitis or peritonsillitis.

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43. Thurley, E. L.: Dangers Associated with Removal of the Tonsils and Adenoid Growths, M. News, New York, March 12, 1904; cited by Ard, F. C.: Dangers Associated with Removal of the Tonsils and Adenoid Growths, M. Rec. **75**:383, 1909.

44. Putnam, cited by Ard, F. C.: Dangers Associated with Removal of the Tonsils and Adenoid Growths, M. Rec. **75**:383, 1909.

45. Hahn, F.: Meningitis nach Angina: Trepanation; Heilung, München. med. Wchnschr. **63**:1822, 1916.

46. Jennemann, F.: Meningitis bei Angina, Ztschr. f. Laryng., Rhin., Otol. **26**: 372, 1936.

47. Siemerling, cited by Jennemann.[46]

48. Fränkel, E.: Ueber postanginose Pyämie, Virchows Arch. f. path. Anat. **254**:639, 1925.

49. Smith, A. B., and Scott, C. E.: Streptococcic Mastoiditis and Meningitis Following a Simple Tonsillectomy with Recovery, J. Laryng. & Otol. **53**:256 (April) 1938.

50. Jacobsen, C.: Complications Attending Operations on Palatal and Pharyngeal Tonsils, Acta oto-laryng. **10**:568-570, 1927.

51. Proskauer, A.: Zur Pathologie des peritonsillären Abscesses, Med. Klin. **10**:1407, 1914; Berl. klin. Wchnschr. **51**:1241, 1914.

52. Harris, S. E., and Yenikomshian, H. A.: Pneumococcus Meningitis Following Tonsillectomy and Terminating in Recovery, Lancet **1**:143 (Jan. 18) 1936.

53. Lewin, R.: Beitrag zur Klinik einiger endokranieller Komplikationen, von Rachen und Mundhöhle ausgehend, Beitr. z. Anat., Physiol., Pathol. u. Therap. d. Ohres **23**:31, 1926.

54. Neumann, H., cited by Beck, O.: Meningitis purulenta nach eitriger Tonsillitis, Wien. klin. Wchnschr. **27**:684, 1914.

55. Reisman, D.: Tonsillitis Associated with Acute Aseptic Meningitis, Ann. Int. Med. **9**:1427 (April) 1936.

56. Frühwald, V.: Ueber phlegmonöse Komplikationen nach Tonsillektomie, Wien. med. Wchnschr. **77**:1027 (July 30) 1927.

EXTRADURAL AND SUBDURAL ABSCESS, ENCEPHALITIS AND BRAIN ABSCESS

Table 4 indicates the incidence of dural infection, acute encephalitis and brain abscess following tonsillar infections. Including our own, there were 13 cases in this series. Eleven patients were males and 2 females. There were 1 patient in the first decade, 4 in the second, none in the third, 4 in the fourth and 4 in the fifth. The youngest was a 7 year old boy and the oldest a 48 year old man. In addition, Parry,[57] Lyons [58] and Vogel each reported a case in which the condition was secondary either to otitis or to mastoiditis. In another case reported by Vogel the disease was rhinologic in origin. In all these cases the intracranial involvement was more or less closely associated with tonsillar infection or operation.

In reviewing these reports one cannot help but he impressed with the statistical errors which have crept into the literature and the loose

TABLE 4.—*Incidence of Dural Abscess, Encephalitis and Brain Abscess of Tonsillar Origin*

	Extradural Abscess	Parapituitary Abscess	Subdural Abscess	Encephalitis	Cerebral Abscess
Tonsillectomy	0	0	1	0	1
Acute tonsillitis	0	0	0	0	1
Peritonsillar abscess	2	1	0	2	6
Total	2	1	1	2	8

manner in which the entire subject has been treated. This noncritical acceptance of uncertain or unverified cases in reviews of the literature gives rise to long series of cases, which leaves the reader with an erroneous impression of the frequency of certain complications. These errors are perhaps not so surprising when one considers (1) the known unwillingness on the part of relatives to permit a complete autopsy, often resulting in lack of adequate evidence for critical study, (2) the imperfectly studied cases, (3) the failure of the average pathologist accurately to describe and localize the lesion within the brain, (4) the injury to brain tissue incident to the removal of the intracranial structures which often obscures the vital steps in the tissue changes, both before and after death and, (5) finally, the fact that to many otolaryngologists the brain is more or less terra incognita.

57. Parry, L. A.: A Case of Acute Mastoiditis with Lateral Sinus Suppuration and Cerebellar Abscess as Complications of the Operation for Removal of Tonsils and Adenoids, Lancet **1**:1821, 1909.

58. Lyons, H. R.: Unusual Complications of Acute Tonsillitis, J. A. M. A. **81**:1605 (Nov. 10) 1923.

RARER COMPLICATIONS

Of the rarer complications reported, cerebral embolism, followed by hemiplegia, heads the list. There were 7 cases of this type, 5 of which followed tonsillectomy and 2 peritonsillar incision. Four patients were males and 2 were females, and the sex of 1 was not mentioned. There were 3 patients in the first decade, 2 in the second and 1 in the fourth, and the age of 1 was not mentioned. The youngest was 32 months and the oldest 32 years of age. One recovered. Two died after tonsillectomy, 1 on the tenth and the other on the sixteenth postoperative day. One had residual facial paralysis, and another had spastic paralysis and was blind and deaf. One, a 32 month old boy, fell back in bed and died within twenty-three hours after the removal of tonsils and adenoids, of pontile hemorrhage. In another patient compression of the medulla by the odontoid apophysis brought about gradual paralysis and death.

AGE AND SEX DISTRIBUTION

Table 5 indicates the age and sex distribution of the cases in this series. It will be noted that 40 of 76 cases, or 52 per cent, occurred in the second and third decades of life. In 51 cases the patients were males and in 20 females, and in 5 no mention was made of the sex; thus the proportion is more than 2 males to 1 female.

PROPHYLAXIS AND TREATMENT

From the preceding discussion it is evident that intracranial complications of tonsillar origin are invariably fraught with grave consequences. Treatment, then, must be essentially preventive, adequately and appropriately applied before infection spreads to the intracranial space. By far the largest number of complications occurred in the form of retrograde thrombophlebitis emanating from the tonsillar veins and involving the cavernous sinus or other dural sinuses. All observers agree that surgical trauma plays an important part in the etiology of thrombophlebitis. This is particularly true in cases of fulminating peritonsillitis in which an incision is made in an attempt to evacuate the abscess, which may or may not be present. Multiple incisions appear to add infinitely to the patient's danger. To operate on an acutely inflamed tonsil before the local tissue immunity is established is only to court disaster. It is a safe rule to defer operative procedure to from four to six weeks after an acute inflammatory attack.

In the acute form of cavernous sinus thrombosis the mortality is 100 per cent. Surgical treatment of well established cavernous sinus thrombosis has been notoriously disappointing. Claus stressed the fact that sharp, piercing pain back of the eye, in the presence of sepsis of tonsillar origin, is indicative of impending thrombosis of the cavernous sinus. He recommended removal of the affected eye, even though the

TABLE 5.—*Age and Sex Incidence of Intracranial Complications of Tonsillar Origin*

Complication	Age Decade								Sex		
	1-10	11-20	21-30	31-40	41-50	51-60	61-70	Unknown	♂	♀	Unknown
Thrombosis of lateral sinus	..	1	..	..	1	1	..	1	0	3	1
Thrombosis of cavernous sinus following tonsillectomy and adenoidectomy	1	1	1	..	3	..	..	1	4	1	2
Thrombosis of cavernous sinus following acute tonsillitis	1	4	2	2	..	..	..	1	9	1	..
Thrombosis of cavernous sinus following peritonsillitis and peritonsillar abscess	2	9	9	2	2	1	1	..	20	6	..
Meningitis	1	4	3	..	..	..	..	1	3	5	1
Extradural and subdural abscess, encephalitis and brain abscess	1	4	..	4	4	..	..	..	11	2	..
Rarer forms	3	2	..	1	..	..	..	1	4	2	1
Total	9	25	15	9	10	2	1	5	51	20	5

ocular examination reveals nothing abnormal. This probably applies only to cases in which the ophthalmic veins are primarily involved and offers no solution in cases in which the infection passes the pterygoid plexus or other venous tracts at the base of the skull.

Successful surgical approach for the management of cervical abscess of whatever origin is now within the realm of possibility. During the course of acute angina or after the operation on the tonsils, if trismus and sepsis continue one should think of the possibility of thrombophlebitis of the internal jugular vein. Obliteration of the subangular space in conjunction with the aforementioned signs is considered a pathognomonic sign of deep-seated abscess in the neck, even though one may not be able to elicit tenderness on palpation on the affected side. In case of doubt the vascular channels should be investigated and a proper surgical measure instituted according to indication before the infection enters the cranium.

Shapiro [59] stated that deep cervical infection is almost exclusively a complication of tonsillectomy which has been performed with local anesthesia. He contended that the contaminated instruments and contaminated anesthetic solution are the primary factors in introducing the infection into the peritonsillar tissue and that at times lack of aseptic technic during the operative procedure may introduce virulent organisms into the blood stream. When freshly made, sterile anesthetic solution or ampules of anesthetic solution, prepared and marketed by reliable pharmaceutic firms, are used, the danger from this source can be greatly reduced.

Of the 7 cases of thrombosis of the cavernous sinus following the operation on tonsils and adenoids in our series, general anesthesia was used in 1 and local anesthesia, presumably, in 5, and in 1 case the type of anesthetic was not mentioned. There is no evidence in these cases to support the contention that the anesthetic agent had anything to do, directly or indirectly, with the causation of the thrombophlebitic process in the cavernous sinus. Of the 9 cases of verified meningitis reviewed in this study, 1 followed adenoidectomy and 8 adenotonsillectomy, in all of which general anesthesia was used.

SUMMARY AND CONCLUSIONS

Intracranial complications of tonsillar origin are extremely rare, only 1 instance (1 of the 2 cases herein reported) being found in an autopsy series of 25,000 cases. In a considerable number of the cases reported in the literature the patients recovered from their lesions, which were

59. Shapiro, S. L.: Deep Cervical Infection Following Tonsillectomy: Report of Three Hundred Cases with Review of Literature, Arch. Otolaryng. **11**:701 (June) 1930.

obviously benign and the nature of which was often unverified. Operative trauma on recent lesions seems to play an important role in the etiology of such complications.

Two cases of intracranial complications of acute tonsillar infection are herein reported. In the first case cellulitis of the neck and then of the scalp developed, followed by thrombosis of the superior longitudinal sinus and an abscess of the brain. In the second case surgical drainage of a peritonsillar abscess was followed by repeated profuse hemorrhages from the nasopharynx and ultimately by thrombosis of the middle cerebral artery. In both cases the lesions were verified at necropsy.

A survey of the available literature since 1900 discloses the fact that most of the intracranial complications of tonsillar origin occur in the form of sinus thrombosis or septic meningitis. Thrombosis of the venous sinuses seems usually to follow more or less direct and consecutive thrombosis of the jugular vein (to the lateral sinus) or of the pharyngeal or pterygoid and associated plexuses (leading to the cavernous sinus). It is quite possible that infection in cases of septic meningitis follows these same routes in some cases; in other cases the lesion is probably a consequence of bacteremia. Lesions within the brain substance proved to be of direct tonsillar origin are extremely rare. While some complications are entirely unavoidable, withholding surgical trauma from acute lesions will probably prevent many of them.

436 South Boyle Avenue.

401 Bailey Street.

RÉSUMÉ OF REPORTED CASES

I. Thrombosis of Transverse Sinus (4 cases)

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1903	Banti, cited by Malan	45	F	Repeated jacksonian convulsions, following acute cold; right hemiparesis and dysarthria; no ocular disturbance.	Suppurative tonsillitis with staphylococci, which brought on thrombosis of right jugular vein and lateral sinus over the torcular to within inferior longitudinal sinus and terminal segment of vena magna of Galen.
1910	Bouvier	58	F	Left tonsillar abscess; chronic tonsillitis (right); induration of left side of neck. Mastoid opened but normal.	No meningitis; thrombophlebitis of left transverse sinus; spread by thrombosis of left jugular vein.
1930	Shapiro			Tonsillectomy, followed by phlegmon of neck.	Thrombosis of jugular and lateral sinus.
1930	Dixon and Helwig	18	F	Tonsillitis (left) for 9 days.	Thrombosis of internal jugular vein, jugular bulb and lateral sinus; culture yielded streptococci.

II. Thrombosis of Cavernous Sinus

A. FOLLOWING OPERATION ON TONSILS AND ADENOIDS (7 CASES)

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1910	Dean	13	M	Tonsillotomy under local anesthesia. Patient left hospital following day, without surgeon's consent, and rode bicycle 8 miles home; a week later septic phlebitis developed, involving internal jugular vein with extension to jugular bulb, then cavernous sinus, terminating in thrombosis of orbital and ophthalmic veins. Well marked right optic neuritis and left exophthalmos, with panophthalmitis. Left eye later required enucleation.	
1913	O'Malley cited by Davis			Course 3 wk.	Osteomyelitis of body of sphenoid bone, which involved cavernous sinus by direct spread along optic nerve on one side.
1920	Chisolm and Watkins	25	M	A soldier had diphtheria bacilli in throat, found during routine examination; infection did not clear up in a month in isolation ward. Tonsils removed. On 3d postoperative day frontal headache and fever developed, and on 6th day cavernous sinus thrombosis, first on right. Died 2 days later.	Basilar meningitis.
1928	Claus	31	M	Frequent sore throat in childhood. Sore throat on Nov. 27, 1927, did not recover; admission Dec. 21 following 8 chills; adenopathy near right angle of jaw; both anterior and posterior common facial veins ligated on day of admission to hospital and right jugular vein encircled by silk suture above and below, but not tied until Dec. 25, because of rise in temperature. On Dec. 30 bilateral tonsillectomy. Veins of left capsule thrombosed. On Jan. 1 slight paresis of abducens and exophthalmos and stiff neck; then signs of thrombosis of cavernous sinus.	Pus above ligature of jugular vein; thrombosis of right inferior ophthalmic vein and right and left lateral sinus; veins of left orbit free.
1928	Claus	31	M	Sore throat; painful swallowing; high fever; vomiting; 3 chills in 4 days before admission. Submaxillary adenopathy on left. Left internal jugular vein ligated above and below, also facial vein anteriorly and posteriorly; tonsillectomy done at same time. Facial vein thrombosed in part. Pronounced icterus and pathologic signs	Thrombosis of internal jugular vein; pus in left pterygoid fossa, which extended to left cavernous sinus; thrombosis of pial vein; toxic reaction of lungs, heart, pleura, kidneys, spleen and liver.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1928	Claus	31	M	in base of left lung posteriorly; next day rales at base of right lung posteriorly; narrowing of left palpebral fissure; on Nov. 11 rales spreading; swelling of the left knee. Patient died Nov. 12.	
1929 1931	Ide Comer	10	M	Operation at meeting of Arizona State Medical Association. Normal convalescence until 3d day, when swelling was noted under and about angle of jaw. Cellulitis continued to spread into neck and face. By 5th day right eye became proptosed and fixed, followed by similar involvement of left eye on 6th day. No blood culture.	Autopsy not permitted.
1932	Campbell	35	F	On day following tonsillectomy right peritonsillar abscess developed. Incised and drained. In 72 hr. thrombosis of cavernous sinus developed. Blood culture showed nonhemolytic streptococci.	Multiple metastatic abscesses; positive blood culture. Head not opened.
				B. WITH TONSILLITIS (11 CASES)	
1907	Seggel, cited by Dean	20	M	Robust soldier; exophthalmos of right eye, with headache, chills, vomiting and intermittent fever and violent phlegmon of right tonsil; blindness and immobility of right pupil, followed by same symptoms on left except there was no exophthalmos; two days after exophthalmos appeared right jugular vein was felt as hard cord. With mercury inunctions the patient recovered within 2½ months, with atrophy of both optic nerves. Total blindness on right and partial on left.	
1909	Lewis	12	M	Four days after onset, pain on left side of neck, left eye and right side of chest; temperature 104 F.; ecchymosis of conjunctiva of left eye and eyelid; marked exophthalmos and paralysis of left side of face; purulent exudate covering left tonsil. Patient talked rationally when spoken to but was somewhat drowsy.	Bilateral thrombosis of cavernous sinus, inferior petrosal sinus and orbital vein and orbital cellulitis; septic infarct of lung; pyemia.
1919	Simeon, cited by Bouvier	Not stated	M	Onset with sore throat and fever; exophthalmos and chemosis; frontal sinus opened but free from infection. Death 3 days after admission.	Upper part of right cavernous sinus contained pus, lower part coated with pus; right internal jugular vein thrombosed as far down as bifurcation; orbital cellulitis, purulent basimeningitis.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1913	Davis	10	M	Healthy child sick one week with follicular tonsillitis on left. Incision made in palate, tonsil, neck and eyelids but no pus obtained. In 10 days well developed cavernous sinus thrombosis; twitching and paresis of leg. Fatal termination in 18 days.	No autopsy; assumed that infection passed into cranium through ophthalmic vein.
1920	Chisolm and Watkins	32	M	Fracture of skull 2 years before. Felt bad for 2 months; then had sudden headache and pain over left eye, nausea and chill; later had swelling in left temple and cheek. Before admission had left mastoidectomy, without finding lesion. Mentally dull and irrational. Profuse sweating of right side of body; right tonsil enlarged; white blood count 32,000; spinal fluid showed Staphylococcus aureus. Day following simple mastoidectomy had double exophthalmos; pupils dilated; no choked disk. Tracheotomy done but patient died.	Thrombosis of both cavernous and lateral sinuses; empyema of left antrum; pus extending along mandibular branch of 5th nerve, through foramen ovale to left cavernous sinus; abscess and necrosis of posterior wall of pharynx and meningitis due to Staph. aureus.
1926	Fränkel	32	M	Pain on right side of throat for 3 days and patient severely ill; marked pressure pain along cervical glands. Angina no longer present. Ligation of right jugular vein. Increasing icterus and meningitis.	Pus in right cavernous sinus; purulent infiltration of soft meninges in middle cranial fossa; same in meninges in right fossa of Sylvius and in cisterna cerebellomedullaris.
1927	Podesta	17	M	Sore throat and fever; pain in left side of neck; pronounced left exophthalmos; headache. In 9 days spinal fluid turbid; blood culture negative. Mastoid explored but no lesion found. Coma and death 11 days after onset.	Subarachnoid fluid turbid; basal leptomeningitis of temporosphenoidal lobe; thrombosis of left cavernous sinus extending into left ophthalmic vein and coronary sinus; right sinus normal; abscess of retropharyngeal wall; toxic reaction.
1935	Ameriso	19	M	Tonsillitis of 6-8 days' duration; patient unconscious when admitted to hospital; marked exophthalmos, worse on left. Exploration of left tonsillar fossa; no pus.	Thrombosis of cavernous sinus; pericavernous edema; septic meningitis; septic infarct of hypophysis; thrombosis of internal jugular vein; pus in sphenoid sinus. Route: pterygoid plexus.
1936	von Kecht	6	F	Purulent otitis on left for 4 days; throat congested, tonsils large. Day after admission meningitis developed. Spinal fluid positive for pneumococcus.	Basilar meningitis; edema of brain; thrombosis of right cavernous sinus; tonsillitis, with deep pus pocket. Ears normal.
1938	Filipo	22	F	Streptococcic tonsillitis (right); right peritonsillar area opened but no pus obtained; right side of neck very painful on palpation; blood culture positive for streptococci. Death with phenomena of cardiac adynamia.	Extensive thrombosis of superficial petrosal and lateral sinuses and of internal jugular vein and hemorrhagic serous meningitis; multiple metastatic pulmonary abscesses.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1939	Witchell	28	M	Acute tonsillitis and retropharyngeal abscess on left. Incision and drainage established on the 15th day. Organism was nonhemolytic streptococcus. Several days of septic temperature followed incision of abscess. Exploration of left side of neck failed to reveal any extension along internal jugular vein. Repeated blood cultures negative. Aphasia developed on 22d day of illness; then abscess of brain considered.	Extensive subdural abscess over temporofrontal area; thrombosis of left cerebral venous sinus. Route: Pterygoid plexus in foramen ovale and foramen of Vesalius. From cavernous sinus infection spread into the superior petrosal and torcular.
				C. WITH PERITONSILLITIS (26 CASES)	
1903	Tollens	19	F	Sore throat, headache and fever; right half of soft palate and postpharyngeal wall swollen. Repeated incision in right tonsillar region finally gave pus, which on culture yielded streptococci; proptosis and meningitis developed.	Dura tense; cavernous sinus and orbital vein thrombosed; right gasserian ganglion covered with pus. Suppuration followed fine veins in pharyngeal plexus to cavernous sinus, outlet of which was filled with pus.
1903	Jessop	31	M	Recurrent quinsy 3 weeks before admission. Neck tender under left side of jaw but no facial paralysis; left eye proptosed. Swelling behind palate incised but watery fluid only evacuated. Became rapidly comatose. Death in 16 days.	Purulent meningitis; right temporal lobe softened; body of sphenoid bone suppurating; both cavernous sinuses purulent; nasal sinuses free; abscess between internal and external pterygoid muscle.
1908	Jacques and Lucien	46	M	Right peritonsillar abscess incised by galvanocautery too early; next day opening enlarged and great quantity of pus obtained, without improvement. On 5th day patient became delirious; septicemia and death.	Thrombosis of cavernous sinus and coronary sinuses; osteomyelitis of the sella; middle ear and paranasal sinuses normal. Route of infection through parapharyngeal venous plexus, which macroscopically was not changed.
1910	Cohen, cited by Bouvier	20	F	Acute sore throat; odynphagia and fever; radiating pain in neck and occiput; submaxillary glands swollen and several days later swelling in floor of mouth; still later cyanosis and death.	Abscess in deep structures of neck; necrosis of sphenoid bone by extension; thrombosis of left cavernous sinus; air cavities free; multiple abscesses in lung.
1912	Long	28	M	Incision for streptococcic quinsy (left); ½ ounce (15 cc.) of green pus obtained; four days later edema and ecchymosis of left eye; temperature septic and delirium most of time. Internal jugular vein ligated. Left eye became blind. Neuritis of third, fourth and sixth cranial nerves from extension along inferior or superior petrosal nerve. Recovery.	

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1917	Gott	22	M	Onset with tonsillitis on left side; spontaneous rupture of abscess; edema of temporal region; chemosis of left eyelid; subconjunctival edema and exophthalmos; anuria and Cheyne-Stokes respiration. Death.	No autopsy.
1919	Wylie and Wingrave	48	F	Neglected right peritonsillar abscess incised and drained; signs of cavernous sinus thrombosis first began on left and later affected right side, accompanied by bilateral exophthalmos; patient became unconscious in 3 days and remained so until death, nearly a month after onset.	Extensive necrosis of parapharyngeal structures; cortical abscess of right temporal lobe caused by extension from base of skull; pus filling all ventricles; thrombosis of jugular bulb, sigmoid and lateral sinuses; thoracic and abdominal viscera not involved. Death from pyemia.
1921	Kernan	18	M	Left peritonsillar abscess incised twice, 16 and 8 days before admission. Patient extremely toxic. Signs of thrombosis of left internal jugular vein. Temperature 108 F. on morning after admission. Patient became unconscious and died 20 hr. after hospitalization.	Meningitis and abscess of left temporal lobe; multiple abscesses in lungs and beginning of endocarditis; abscess in left pharyngomaxillary space. Route: Secondary to thrombosis of lateral sinus. Thrombosis of cavernous sinus, lateral superior and inferior jugular veins; mastoid normal.
1921	Hösten	20	M	Some improvement following incision for quinsy, followed by chills, exophthmos and coma. Death in 5 days.	Necrosis of both sphenoid and occipital bones, in addition to thrombosis of cavernous sinus and basilar meningitis. Route of infection: not stated.
1923	Reye	24	M	Onset with odynphagia and high fever because of quinsy. Tenderness of right sternocleidomastoid muscle. Right jugular vein ligated. Persistent septic fever; exophthalmos and death.	Retropharyngeal abscess; putrid thrombosis of right jugular vein; bilateral thrombosis of cavernous sinus and meningitis. Metastatic pulmonary and multiple periarticular abscesses.
1925	Meslin and le Barazer	13	M	Recurrent tonsillitis; violent headache and fever. Exploratory mastoidectomy negative. Temperature continued septic; left peritonsillar and parapharyngeal abscesses drained. Pronounced exophthalmos. Both orbits successfully drained but patient died of exhaustion.	No autopsy. Route: Not stated. Metastatic pyemia with pulmonary abscess.
1925	Ufflenorde	20	M	Peritonsillar abscess; cellulitus of floor of mouth and swelling of submaxillary glands.	Infection spread by parapharyngeal space, causing thrombosis of cavernous sinuses. Secondary metastatic abscess of lung.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1926	Pollak	30	M	History of previous peritonsillitis. Present illness of 26 days' duration, with septic temperature and chills. Right half of face greatly swollen. Rapid decline, coma and death.	Thrombosis of right internal jugular vein; brain soft and edematous; suppurative meningitis. Spread by contiguous dissemination to endocranium.
1926	Rössle	21	M	Peritonsillitis of left side of 8 days' duration.	Basal meningitis and encephalitis; acute regional lymphangitis; suppuration of cavernous sinus; phlegmon of left orbit. Metastasis to the lungs; pleuritis; mediastinitis. Serial section showed phlegmon of parapharyngeal space and phlebitis of pterygoid and pharyngeal plexuses. Culture revealed Str. viridans and influenza bacilli.
1929	Schlittler	22	M	Six days before admission pain with swallowing, headache, fever and sensation of tension in left side of throat. Repeated chills. Peritonsillitis on left side; trismus; stiffness of neck; paralysis of oculomotor and abducens nerves; chemosis of left upper eyelid. Death on 9th day.	Purulent phlebitis of left pharyngeal vein; basal meningitis and suppuration of left cavernous sinus; phlegmon of left orbit. Hemorrhagic purulent metastasis of both lungs, pleura and mediastinitis. Str. viridans on culture. Route: Probably pterygoid plexus.
1930	Dixon and Helwig	19	F	Incision on right. Fourteen days after onset, proptosis of right eye, which in 2 days receded as suddenly as it appeared; delirium. Next day severe chest pain. Left eye never involved.	Embolic abscesses in lungs and heart; parapituitary abscess. "Primary involvement of the right cavernous sinus found its origin from the tonsillar region by way of the internal jugular vein."
1930	Malan	25	M	Bilateral peritonsillar abscess, first on right and then on left. Both drained. Trismus and swelling of left side of face. Later parapharyngeal space explored and abscess evacuated. Sepsis continued. Bilateral exophthalmos, coma and death.	Basal meningitis and encephalitis; gray matter apparently normal; pus in 4th ventricle, which invaded lower portion of sylvian duct; left cavernous sinus and pterygoid plexus thrombosed; small extradural abscess at confluence with ophthalmic vein.
1931	Wessely	18	M	History of quinsy a year before and pleurisy 6 wk. before present illness. Peritonsillitis on left of 10 days' duration. Incision and drainage of abscess. Swelling over left temporal region. Tonsillectomy followed by cervical cellulitis but no pus on incision; internal jugular vein ligated. Progressive swelling of both eyeballs. Coma and death.	Dura tense; meninges edematous; subdural abscess; gyri flattened; consistency of brain reduced. On section tissue damp; ventricles slightly distended; fluid cloudy and yellowish; both cavernous sinuses thrombosed; pharyngeal tissue edematous and abscess cavities in the muscles.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1931	Turner and Reynolds	34	F	Spontaneous rupture 1 wk. after onset; two weeks after, patient was hospitalized, with signs of thrombosis of cavernous sinus. Both upper and lower eyelids swollen and left eyeball proptosed; patient became unconscious and died 3 days after admission.	Meningitis extending into tentorium cerebelli and posterior cistern; both cavernous sinuses containing pus; pyemic abscess in each lung.
1931	Wessely	51	M	History of repeated quinsy since childhood. Onset with violent pain in left side of throat; swelling of the left side of face; trismus. Patient refused incision but improved temporarily. A month after onset complete ophthalmoplegia and left chemosis developed. Death followed exploration of air cavities.	Abscess in left pterygoid muscle; thrombosis of both cavernous sinuses; necrosis of dura. Route: Spread by direct continuity from pharynx into central nervous system.
1931	Wessely	55	M	Soreness of left side of throat; septic temperature; trismus, and swelling of left side of neck, which a week later spread to left temporal region, with edema of both eyelids.	Protrusion of both eyelids. Pus in pterygoid muscles; thrombosis of pterygoid plexus; extradural abscess; cavernous sinus filled with pus. Route: Direct phlegmonous extension.
1933	Vogel	10	M	Swelling of right side of face associated with peritonsillitis; thrombophlebitis of internal jugular and facial veins, requiring excision of a segment. Death 9 days after onset.	Streptococcic sepsis coming from a branch of the facial vein and the cavernous sinus and superficial cervical veins; large retrotonsillar abscess; edema of larynx; lymphangitis of right internal carotid sheath; cortical abscess of right temporal lobe; parenchymatous change of internal organs; pus coating right 8th nerve.
1934	Claus	21	M	Signs of thrombosis of cavernous sinus on 8th day of illness following evacuation of pus from peritonsillar abscess; complaint of piercing pain of one eye. Immediate exenteration of affected eye, followed by recovery.	
1936	von Kecht	61	M	Sore throat for 3 days, then left facial paralysis; cheek swollen and parotid gland tender. Spinal fluid showed gram-negative cocci. External drainage of parapharyngeal space.	Purulent basilar meningitis; thrombosis of left cavernous sinus, superior sphenoid and sigmoid sinuses. Route: Parapharyngeal space, thence to parotid gland and pterygoid plexus before invading the intracranial structures.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1936	von Kecht	17	M	Left peritonsillar abscess for 8 days; left side of face swollen and submaxillary glands much enlarged; signs of meningitis; left eye ecchymosed. Spinal fluid milky but culture sterile. Tonsillectomy and mediastinotomy. No pus. Internal jugular vein ligated. Meningeal signs increased next day and patient died following day.	Infection spread from phlegmonous tonsil on left to pterygoid muscle. Suppurative cavernous sinus, meningitis and encephalitis. Parenchymatous degeneration of internal organs.
1936	Schubel, cited by Jennemann	2	F	Appendectomy and 2 mastoidectomies performed on basis of mistaken diagnosis. Tonsillogenic abscess ruptured through anterior auditory canal. Right tonsillectomy. External drainage of right parapharyngeal space. Death.	Peritonsillitis on right; rupture into external auditory canal and parotid gland. Death from meningitis.
				III. Verified Meningitis (9 cases)	
1912	Levy	11	M	Adenoidectomy and death in 2 wk.	Diffuse purulent meningitis, predominating over convexity on right; no adhesion of dura or involvement in region of left petrous bone or basilar portion of occipital bone.
1914	Beck	7	F	Peritonsillitis overlooked. Extensive destruction of base of skull from symptomless retropharyngeal abscess. Streptococci in spinal fluid.	Purulent basilar meningitis; bilateral suppurative tonsillitis, from which phlegmonous infection spread toward dura.
1919	Wachter	17	F	Tonsillitis 3 wk. after institution of treatment for thyroiditis, followed by signs of meningitis; spinal fluid yielded Staph. aureus.	Incomplete autopsy. Dura and pia under increased pressure; purulent meningitis; infectious thrombophlebitis, thought to have spread from tonsillar region.
1924	Wessely	29	M	Repeated incisions for peritonsillitis. Cervical cellulitis. Mediastinotomy; resection of internal jugular vein, but no abscess found. Death from sepsis.	Suppurative meningitis; phlegmonous anterior mediastinitis; marked cellulitis of tonsils and neck.
1929	Gunther	12	M	Frequent sore throat for 3 yr. Tonsillectomy with local anesthesia, followed by retropharyngeal abscess, involving cervical vertebra.	Purulent osteomyelitis of epistropheus, with perforation of dura; purulent meningitis of base and convexity of cerebrum.
1930	Shapiro	Adult		Local tonsillectomy.	Basilar meningitis; pus in carotid sheath and spread of infection to meninges.
1930	Dixon and Helwig	30	F	Bilateral phlegmonous tonsillitis, followed by cervical abscess. Death in 10 days.	Numerous hemorrhages in dura at base and purulent meningitis.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1936	Harris and Yenikomshien	18	F	Local tonsillectomy. On 6th postoperative day severe headache and fever; on 10th day spinal fluid became turbid; culture yielded pneumococci, not type III. Repeated lumbar puncture and administration of intrathecal polyvalent antipneumococcus serum.	Recovery in a month.
1936	von Kecht	30	F	Acute tonsillitis of 3 days' duration, followed by signs of meningitis. Lumbar puncture yielded Str. haemolyticus.	Purulent meningitis; cerebral edema; cervical abscess, and cellulitis of tonsils.
	IV. Primary Encephalitis and Brain Abscess (11 cases)				
1907	Kandler, cited by Wessely	35	F	Patient refused incision of left peritonsillar abscess until meningeal symptoms occurred. Death in 8 days.	Abscess of left temporal lobe; encephalitis; abscess from preauricular site and retropharyngeal abscess. Pus penetrated wall of pharynx and extended along fibers of stylopharyngeus muscle, next through connective tissue, along which route suppuration extended to skull and entered through sphenopetrosal fissure. Pneumonia and pleurisy.
1927	Ferretti	40	M	Acute tonsillitis and edema of larynx for 2 wk., followed by peritonsillar abscess. Antisyphilitic treatment. Four hemorrhages from tonsillar wound; hemiparesis; abscess of the inferior portion of the prerolandic and postrolandic areas drained 50 days after onset of illness.	No autopsy. Speculative diagnosis: Infection of lateral pharyngeal lymphatics, deep cervical lymph nodes and ascending endocranial lymphatics in perivascular sheath of sylvian artery.
1929	Housden	18	M	Acute tonsillitis for 2 mo.; in 10 days signs of cerebral abscess or meningitis; jacksonian convulsions in 22 days. Coma and death 2 mo. after onset.	Partial autopsy. Extension of abscess from area of osteomyelitis in greater wing of sphenoid; encapsulated abscess of temporal lobe, with adhesion.
1929	Goodyear	18	M	Right peritonsillar abscess incised 3 wk. before examination. Right side of face partly paralyzed; right corneal reflex absent. Right side of neck incised on admission. Two days later right eyelid and upper part of orbit swollen; area incised but no pus. Right hemiplegia 13 days after entry.	Parapituitary abscess 3 cm. wide and 1 cm. deep; right side of cerebellum covered with pus; cavernous and other sinuses not involved; thrombosis of external jugular vein and normal internal jugular vein.
1930	Johnson	7	M	Removal of tonsils and adenoids 12 days after acute tonsillitis. Five days later fever and chills; on 10th postoperative day left hemiplegia, followed by coma and death on 12th day.	Diffuse meningitis and encephalitis; bronchopneumonia and metastatic abscess anterior to fissure of Rolando on right, according to illustration.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
1930	Cadbury and Siddall	41	M	Tonsillectomy under procaine infiltration anesthesia followed by jacksonian convulsions on 5th postoperative day. Death 20 days after operation.	Multiple abscesses of right frontal, temporal and parietal lobes. Impression of authors: Distribution of abscesses indicates they were due to direct transmission of septic material from right tonsil by needle puncture through internal carotid artery.
1930	Dixon and Helwig	13	F	Severe tonsillitis for 5 wk., then right peritonsillar abscess, followed by left hemiplegia, associated with repeated uncontrollable tonsillar hemorrhages from original incision 14 days after admission.	Encephalomalacia over right temporoparietal region, with acute vascular hyperemia and breakdown of brain substance. Death from exsanguination.
1931	Eliasson	45	M	Onset with toothache. Trismus. Peritonsillar abscess following chronic tonsillitis. Abscess opened. Patient became unconscious 12 days later; Kernig's sign positive. Died next day.	Much pus in foramen ovale and foramen lacerum; encephalitis; brain abscess (free) immediately inside and somewhat below the splenium corporis callosi.
1933	Silverberg	45	M	Peritonsillar and retropharyngeal abscess for 1 wk. Peritonsillar abscess, and next day retropharyngeal abscess drained. In 1 wk. right posterior cervical abscess incised along posterior border of sternocleidomastoid muscle, followed by profuse bleeding. Submaxillary region opened but no pus. On 44th day of hospitalization bronchopneumonia, convulsions and left hemiplegia developed, and patient died.	Osteomyelitis of base of skull and occipital bone; extradural abscess; suppurative inflammation of cervical tissue and terminal pneumonia. Direct extension of infection through parapharyngeal space.
1934	Grabscheid	34	M	A year after tonsillectomy cellulitis of left side of pharynx and left cervical abscess developed. Ten days after onset of cervical cellulitis small, localized abscess opened. In 2 days parapharyngeal space opened and irrigated daily. Intracranial complications 2½ mo. later. Exploration of frontal lobe. Culture yielded nonhemolytic streptococci. Death in 15 days.	Subdural abscess and pachymeningitis; parenchymatous degeneration of heart, kidneys and liver; peritonsillar tissue normal. Infection spread by direct extension from parapharyngeal space.
1938	von Soubiron and Emiliani	34	M	Two weeks after tonsillectomy common carotid artery ligated to control hemorrhage in ulceration of unrecognized parapharyngeal abscess and necrosis of external carotid artery. Temporary aphasia and hemiparesis. Cervical abscess extended down to retroclavicular region; abscess controlled by repeated aspiration. Temperature continued high and septic. Jacksonian convulsions 3 days before death.	Cortical encephalitis and brain abscess of left superoposterior portion of parietal lobe; left hemisphere edematous and right one compressed; multiple abscesses in parietal and large central abscess in occipital lobe.

Date	Author	Age, Yr.	Sex	Symptoms and Course	Autopsy Observations
				V. Embolism and Rarer Forms (7 cases)	
1917	Gracey	17	M	Tonsillectomy with ether anesthesia. On 6th postoperative day hemiplegia developed. Spinal fluid culture sterile; cell count between 111 and 181. Numerous jacksonian convulsions.	No autopsy. Cause probably embolism.
1917	Scruton	11	F	Tonsillectomy by a tyro. Left hemiplegia first noticed next evening. Recovery from hemiplegia began 3 days later and was complete in 2 wk. except for residual facial paralysis; probably due to embolism.	
1925	Bailey	32	M	Tonsillectomy under general anesthesia. Dysphagia developed 14 days later, followed by paralysis of right side of face and left half of body.	No autopsy. Possible causes of death: (1) botulism, (2) embolism of brain and (3) infantile paralysis.
1926	Tawse	32 months	M	Hemorrhage within 23 hr. after operation on tonsils and adenoids. Patient died suddenly, after becoming unconscious.	Hemorrhage, size of large peanut, in center of pons.
1926	Woodson	7	M	Repeated attacks of tonsillitis. On 4th day of attack had peritonsillits but no pus; became progressively comatose and remained so until 3d week of illness; recovered. Spastic paralysis developed on left and patient became blind and deaf.	
1932	Apert, cited by Baratoux			Peritonsillitis. Trismus, torticollis and pain in nape of neck. Gradually developing paralysis.	Odontoid apophysis bulged through occipital foramen, compressing meduila against posterior arch and atlas.
1938	Gareiso and Escardo	4	F	Hematemesis, melena and pain in right leg 12 days after tonsillectomy. Next day patient unable to stand or hold objects; active movement observed on left side but diminished on right; right-sided pyramidalism with Babinski and Oppenheim signs on left. Recovery. Three possible causes: (1) embolism, (2) encephalitis and (3) large hemorrhage, as described by Worms.	

GIANT LYMPH FOLLICLE HYPERPLASIA OF THE NASOPHARYNX

REVIEW OF THE LITERATURE; REPORT OF A CASE

J. C. PEELE, M.D.

HAROLD KIMATA, M.D.

KOTZ ALLEN, M.D.

AND

JOHN HALLARON, M.D.

NEW ORLEANS

In 1925 Brill, Baehr and Rosenthal [1] described a previously unrecognized disease entity characterized by generalized lymphadenopathy and splenomegaly and associated with a normal blood picture. The diseased structures exhibited a rapid and complete response to roentgenotherapy, although the authors at this time were unable to say whether the response was only temporary or was permanent. Histologic study revealed giant lymph follicle hyperplasia of the lymph nodes associated with a similar pathologic process in the malpighian bodies of the spleen, with resultant enlargement of these structures. The disease was considered by the authors to be benign. Attention had first been called to this condition by the examination of a lymph node and spleen removed from a patient who died following splenectomy in 1914. The observation of 2 cases in which the condition was similar subsequently led the authors to report these and to attempt to establish this condition as a distinct and unique clinicopathologic syndrome, which they called splenomegalia lymphatica hyperplastica.

Brill and his associates were quick to acknowledge the priority of Becker [2] in the clinical and pathologic description of this condition, although it seems likely from a review of Becker's article that he either did not appreciate the significance of this syndrome or else made no attempt to establish it as a distinct clinical entity. In Becker's case a lymph node removed during life three years subsequent to the onset

From the Department of Otolaryngology of the Eye, Ear, Nose and Throat Hospital.

1. Brill, N. E.; Baehr, G., and Rosenthal, N.: Generalized Giant Lymph Follicle Hyperplasia of Lymph Nodes and Spleen, J. A. M. A. **84**:668 (Feb. 28) 1925.

2. Becker, E.: Contribution to the Study of Lymphomas, Deutsche med. Wchnschr. **27**:726 (Oct. 17) 1901.

of generalized lymphadenopathy and splenomegaly was considered by the pathologist to be "hyperplastic." The blood picture in this case showed a moderate percentage increase in lymphocytes, especially the large forms, with a less marked increase in the total number of leukocytes. The diagnosis was "pseudoleukemia," which, according to Symmers, was a general term employed about this time (1901) to include any unexplained enlargement of the lymph nodes and spleen.

In 1927 Baehr and Rosenthal [3] reported 6 cases of this disease and stated the salient points in differentiating it from lymphosarcoma. They pointed out the fact that it did not involve the tonsils or the lymphatic apparatus of the gastrointestinal tract. For want of a better name they called this condition malignant lymph follicle hyperplasia.

In 1927 Symmers [4] reported 3 cases of a similar nature which had come to his attention and pointed out that the disease could be differentiated from leukemia by the blood picture and from Hodgkin's disease and lymphosarcoma by the histologic architecture. He stated that because of the ready susceptibility to roentgen therapy the condition was benign and the prognosis good. All 3 cases reported presented the same general type of clinical and histologic picture in that clinically there was generalized enlargement of the lymph nodes attended by splenomegaly and microscopically almost exclusively a numerical and dimensional hyperplasia of the germinal follicles of the lymph nodes and malpighian bodies of the spleen.

In 1927 Baker [5] added another report to the slowly mounting list of recorded cases. The diagnosis of giant lymph follicle hyperplasia was made by Dr. H. S. Martland from the study of a gland removed from the postauricular region. The diagnosis was concurred in by Dr. George Baehr, who examined microscopic sections of the gland removed for biopsy. As in other cases of similar nature, there was ready response to irradiation, and the blood was normal. The terminal course in this case is interesting and will be commented on later.

In 1929 Terplan [6] selected for report an interesting case from a large group of cases of lymphogranuloma and other conditions closely related to Hodgkin's disease, reports of which had been published previously.

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3. Baehr, G., and Rosenthal, N.: Malignant Lymph Follicle Hyperplasia of Spleen and Lymph Nodes, Am. J. Path. **3**:550 (April) 1927.

4. Symmers, D.: Follicular Lymphadenopathy with Splenomegaly, Arch. Path. **3**:816 (May) 1927.

5. Baker, R. D.: Splenomegalia Lymphatica Hyperplastica, or Generalized Giant Lymph Follicle Hyperplasia of Lymph Nodes and Spleen, J. A. M. A. **88**: 2035 (June 25) 1927.

6. Terplan, K.: On a Peculiar Systemic Disease Resembling Granuloma, Verhandl. d. deutsch. path. Gesellsch. **24**:65, 1929.

This case was at first regarded clinically and anatomically as one of lymphogranulomatosis, but histologic study of the lymph nodes, liver, spleen and bone marrow showed unusual deviations from the classic picture of the Paltauf-Sternberg granuloma. The author stated the belief that he had described a systemic disease which was previously unrecorded. He apparently was not aware of the previous publications of Becker and the American authors Brill, Baehr, Symmers and Baker.

The first indication that so-called giant lymph follicle hyperplasia of the lymph nodes and spleen might not always pursue the course characteristic of a benign lesion came with the report of Baehr, Klemperer and Rosenthal in 1931.[7] The authors were provided with an opportunity to study sections of lymph nodes and spleens removed from patients, several years subsequent to the time at which the structures were reported to be affected by benign giant lymph follicle hyperplasia.[8] The results of these studies, together with the postmortem observations in some of these cases, served in the opinion of the authors to identify this disease with lymphosarcoma. They concluded, however, that the disease is a form of lymphosarcoma which deserves to be distinguished as a pathologic entity because of its characteristic pathologic changes, its unique evolution and its unusual duration. They reserved for this special form of lymphosarcoma the term "follicular lymphoblastoma." They also pointed out that while the lesion in its early stages is remarkably radiosensitive, the recurrences which eventually take place throughout the course of years become less easily influenced by radiotherapy.

In 1932 Baehr[9] reiterated his conviction that giant lymph follicle hyperplasia of the lymph nodes and spleen was a form of lymphosarcoma. He added further that while the lymph nodes and spleen are most commonly involved, the disease occasionally attacks structures which ordinarily contain little lymphoid tissue, such as the lacrimal gland, the breast, the loose connective tissue of the pelvis, the fat tissue of the orbit and the subcutaneous fat. Nodules of tumor tissue excised from these tissues showed the same histologic picture of characteristic follicle formation. Occasionally the bone may be involved, resulting in pathologic fracture. Of 19 patients periodically observed for some time, 8 were known to have died, while 8 were still alive after periods of observation ranging from one to fourteen years. Three patients of this group could not be traced. The average duration of life for the series was five

7. Baehr, G.; Klemperer, P., and Rosenthal, N.: Follicular Lymphoblastoma (Giant Follicular Hyperplasia of the Lymph Nodes and Spleen), Am. J. Path. **7**:558 (April) 1931.

8. Brill, Baehr and Rosenthal.[1] Baehr and Rosenthal.[3]

9. Baehr, G.: The Clinical and Pathological Picture of Follicular Lymphoblastoma, Tr. A. Am. Physicians **47**:330, 1932.

years and nine months. This is to be contrasted with the usual life expectancy of fifteen months with other types of lymphosarcoma.

In 1938 Symmers,[10] in an excellent article, made some notable contributions to the subject and added some new case reports. He showed clearly that the lesion may become associated with the histologic changes of Hodgkin's disease or of lymphatic leukemia or may undergo direct transformation into polymorphous cell sarcoma or that late necrotic lesions may finally develop in the follicles of the lymph nodes and spleen. He stated the belief that the latter condition was unique and hitherto undescribed. He emphasized the fact that the development of polymorphous cell sarcoma is represented by a transformation of the cells "in situ," whereas the association with Hodgkin's disease is purely indirect, representing a deposition in the modified lymph nodes of megakaryocytes from the bone marrow. It was further stressed that the transformation into polymorphous cell sarcoma is without benefit of mitosis. The relationship to lymphatic leukemia was observed in the case previously reported by Baker [5] as an example of simple giant lymph follicle hyperplasia.

In 1940 Novak [11] reported a case in which a nasopharyngeal tumor was removed surgically, which presented the histologic picture of giant follicular lymph follicle hyperplasia without gross involvement of the lymph nodes and spleen. He stated that he could not find any report of a similar case in which the terminal lymph nodules alone were involved. A similar case was recently brought to our attention, and we have been stimulated to review the literature in an attempt to determine the salient features of this disease, and to report the case under observation.

ETIOLOGY

The cause of giant lymph follicle hyperplasia is not known. The condition has been variously described as "toxic" or "inflammatory" in origin. From our review of recorded case histories this does not seem logical. Generally speaking, the health of the patients has been good until the time of onset of symptoms, and for the most part the major symptoms which developed during the course of the disease were directly or indirectly attributable to the results of compression accruing from the enlarged lymph nodes and spleen. These symptoms were relieved almost "in toto" after the size of these structures had been reduced by roentgen therapy, except, of course, in those cases in which the disease took a malignant course. In only 1 case reported by Symmers [10] were the con-

10. Symmers, D.: Giant Follicular Lymphadenopathy With or Without Splenomegaly, Arch. Path. **26**:603 (Sept.) 1938.

11. Novak, F. J., Jr.: Giant Follicular Hypertrophy of the Nasopharynx: Report of a Case, Ann. Otol., Rhin. & Laryng. **49**:526 (June) 1940.

comitant serologic findings positive. In another case reported by the same author [10] there was associated amebic dysentery, which, however, was considered to have little relationship to the disease in question.

DISTRIBUTION

The structures universally involved have been the superficial and deep lymph nodes, either alone or in combination. Symmers emphasized the fact that the deep lymph nodes may become involved initially. In the majority of cases there was attendant splenomegaly. It is interesting to note that in a case recorded by Symmers the initial lymphadenopathy was in the preauricular region and was bilateral. According to Baehr,[9] structures which ordinarily contain little lymphoid tissue may become the site of disease, as previously mentioned.

PATHOLOGY

The classic histologic description of this condition is that of a numerical and dimensional hyperplasia of the follicles of the lymph nodes and malpighian bodies of the spleen. The germinal centers are composed of large clear endothelioid type cells resembling lymphoblasts, with a dense collection of mature lymphocytes about the periphery of the follicle. Symmers [10] has made some noteworthy contributions to the study of this disease. According to him, the cases can be separated into two groups. The first group is composed of cases in which the hyperplastic condition of the follicles in the nodes and spleen retains the status of simple hyperplasia for months or years; the follicles become smaller or disappear temporarily for no apparent reason, show histologic evidence of healing irrespective of treatment or rupture spontaneously and heal. In the second group he placed those cases in which the disease exhibits more malignant tendencies. We have mentioned this previously but reiterate it here for the sake of emphasis. The follicles undergo a direct transformation into polymorphous cell sarcoma or become associated with the histologic changes of Hodgkin's disease or of lymphatic leukemia or else late necrotic lesions develop in the follicles of the lymph nodes and spleen, a condition which the author considered to be unique and previously undescribed. He also has shown that in some instances the combination of giant follicular lymphadenopathy, polymorphous cell sarcoma and Hodgkin's disease may be seen in the same lymph node. The relationship of this disease to lymphosarcoma was previously pointed out by Baehr.[9]

Symmers stated further that the cells which are derived from the germinal centers of the lymph follicles fall into two main groups and are referred to as large hypochromatic embryonic cells (or shadow forms) or hyperchromatic embryonic cells of the large lymphocytic variety, with forms transitional between the two being discernible. The absence of reticulum cells was demonstrated by special stains.

CLINICAL COURSE

Clinically, this newly recognized form of disease bears a striking resemblance to lymphosarcoma, Hodgkin's disease, lymphatic leukemia and allied diseases. It usually begins with gradual enlargement of the lymph nodes throughout the body, involving the superficial or the deep groups, alone or in combination. In the majority of cases there is associated splenomegaly. The encroachment of the enlarged structures on the surrounding organs gives rise to symptoms of compression which may be referable to the esophagus, tracheobronchial tree, thorax, neck or abdomen. Not infrequently the presence of this abnormal enlargement of structures may not be obvious to the patient. Most commonly the lymph nodes are discrete, movable, firm, painless and not tender, and vary in size from 1 to 5 cm. or more. Spontaneous disappearance of the nodes and recurrence are not infrequent. More rarely, the liver becomes enlarged, and the bones are involved, leading to pathologic fracture. If the disease runs the course of hyperplasia of the purely lymphoid type originally described, the life expectancy is good. The longest duration of life which we have seen recorded under these circumstances was fourteen years. If, however, malignant tendencies are manifested at a later date, the average duration of life is about one to six years from the onset of symptoms.

TREATMENT

The ready response of this disease to roentgen therapy is a highly characteristic feature and almost obviates the necessity of surgical intervention provided the patient is seen when the condition is in a reasonably early stage. While recurrences are common, the tumor masses yield promptly to irradiation. This response is so ready and complete that it has been said that a failure to respond may suggest the possibility of an incorrect diagnosis or a mutation of the simple lymph follicle hyperplasia to a more malignant type.

REPORT OF A CASE

History.—A. P. H., a white man, aged 32, came to the clinic of the Eye, Ear, Nose and Throat Hospital on Jan. 11, 1941 complaining of a "tumor in the throat." The patient first noticed the presence of this mass after the onset of a moderately severe head cold, sore throat and laryngitis about ten days previously. During this time there were the usual attendant general symptoms, together with slight impairment of hearing in the right ear and a feeling of fulness. Shortly after the onset of symptoms, the patient while inspecting his throat noticed the large mass, which persisted after the symptoms of infection in the upper respiratory tract began to wane. Becoming alarmed that this "tumor" might be cancer, he sought treatment in the clinic. The past medical history was essentially noncontributory. In 1937 the patient underwent tonsillectomy in the clinic, because of occasional attacks of sore throat, and since that time he had been relatively free of respiratory symptoms except for scant mucoid postnasal drainage, which

never occasioned any concern. His general health had always been good, and there had never been any serious illnesses or previous operations except tonsillectomy. A history of venereal infection was denied. The family history and social history were not related.

Physical Examination.—The patient presented himself at the clinic with rhinosinusitis, pharyngitis and laryngitis in a subacute stage. Examination after the brief period of acute inflammatory reaction revealed a septal spur posteriorly on the right, which abutted into the posterior tip of the middle turbinate on that side. On nasopharyngoscopic examination a large pinkish gray mass was seen suspended by a broad base from the vault of the nasopharynx, completely obstructing the view of the lateral wall on the right side and filling the right choana. At the vault the tumor mass extended to the left lateral wall of the nasopharynx and down into the fossa of Rosenmüller on the left side, although with this lateral extension to the left it rapidly diminished in size, so that at its termination behind the left torus tubarius it represented a small mass of tissue, which did not, however, assume tumor proportions. On inspecting the pharynx a smooth oval lobulated pinkish gray mass was seen projecting down about ¼ inch (0.64 cm.) below the level of the arch of the soft palate on the right side and extending just to the left of the midline behind the base of the uvula. The mass was firmly

Fig. 1.—Photograph of tumor mass (giant lymph follicle hyperplasia) arising in the nasopharynx and projecting downward into the oropharynx.

adherent to the lateral wall of the pharynx in the region of the fossa of Rosenmüller. It was moderately firm to palpation. The tonsillar fossae were clean. The lingual tonsils were small, and there were a few dilated veins at the base of the tongue. The larynx was normal. There was a moderate degree of thickening and slight retraction of the tympanic membranes bilaterally. A few small lymph nodes were palpable on either side along the anterior borders of the sternocleidomastoid muscles. The heart, lungs and abdomen were clinically normal. The ocular fundi were normal. There was no adenopathy of the axillary, epitrochlear or inguinal regions. The blood pressure was 128 systolic and 80 diastolic. The average weight was 162 pounds (73.5 Kg.). The weight at the time of examination was 160 pounds (72.6 Kg.). Roentgenograms of the chest and paranasal sinuses failed to reveal any pathologic condition. The audiometric reading showed normal hearing except for a 20 decibel loss in the higher tone range on the right side. The Rinne and Gellé tests were positive, and in Weber's test the sound was lateralized to the right. Transillumination of the paranasal sinuses showed slight opacity of both antrums and moderate bilateral opacity of the anterior ethmoid cells.

Laboratory.—The total red blood cell count was 5,360,000, with 80 per cent hemoglobin and a color index of 0.7. The total white blood cell count was 5,250,

with 65 per cent neutrophils, 34 per cent small lymphocytes and 1 per cent basophils. There were 1 per cent myelocytes and 4 per cent each of juvenile and stab forms. The coagulation time of the blood was three minutes and ten seconds. The urine was normal except for a moderate number of pus cells. The Wassermann and Kahn tests of the blood were negative.

Pathologist's Report (Dr. George H. Hauser).—The specimen examined preoperatively was oval in shape and measured about 2 by 6 cm. It was deep gray

Fig. 2.—Photomicrograph showing giant lymph follicle hyperplasia of tumor mass removed from the nasopharynx, with interfollicular fibrosis and chronic inflammatory reaction.

and soft. Microscopic examination of small pieces of tissue removed at operation showed the specimen to be composed of lymphoid tissue with the lymph follicles increased both in size and in number. Some of the follicles were very large. The central portion of each follicle was composed of large cells with pale cytoplasm, while the periphery was made up of thickly packed lymphocytes. There was also a diffuse chronic inflammatory process present, with increase of con-

nective tissue and many cells showing pyknotic and pressed-out spindle-shaped nuclei. No mitotic figures were in evidence.

Operation and Course.—On Jan. 29, 1941 one of us (Kimata), assisted by Drs. Kotz Allen and John Hallaron, with the patient under general anesthesia, exposed the nasopharynx by Beck's method and removed a large nasopharyngeal tumor by means of an adenotome and punch forceps. No difficulties were encountered, and the convalescence was uneventful. The patient was referred to Drs. Leon J. Menville and J. N. Ané for postoperative irradiation. The plan of roentgen therapy as outlined by Dr. Ané consisted of a series of three high voltage treatments, the fields being 10 by 10 cm., over the right and left sides of the neck and face, outlined in such a manner as to cover the nasopharynx. A dose of 134 roentgens (r) was applied to the right and left fields at each visit—a total dose of 804 r. The following physical factors were used: 220 kilovolts, 15 milliamperes and a skin target distance of 50 cm., with 1 mm. of copper plus 1 mm. of aluminum for filtration. The output per minute was 33.5 r. Treatment was started on Feb. 24, 1941 and concluded March 3, 1941.

SUMMARY

In an attempt to determine some of the more significant features of the disease we have reviewed the literature on the subject of giant lymph follicle hyperplasia and have reported a case recently encountered by us in which the pathologic manifestations were strictly limited to the nasopharynx. This case and that first reported by Novak [11] constitute, so far as we know, the only 2 cases of the nature recorded in the literature. Inasmuch as we are not primarily pathologists, we do not feel competent to discuss the varied histologic aspects of lymphoid tumors. It has been apparent as a result of our study that even among competent microscopists a diagnosis of the various tumors involving lymphoid structures frequently cannot be made except through repeated clinicopathologic observations and, often, postmortem observations.

The question of prognosis is paramount with the patient. So far as we have been able to determine there are no clinical or pathologic criteria whereby one is enabled to prognosticate the future course of a tumor definitely diagnosed as purely giant lymph follicle hyperplasia. If one accepts past experience as a guide, then one may say that such a tumor stands a reasonably good chance of progressing to a fatal issue within the relatively brief period of a few years. We know of no more painful duty than to inform a patient that his life span may be shortened prematurely as a consequence of malignant disease.

The relative immunity of the lymphoid tissue of the pharynx to the ravages of this disease is unique and a point of special interest to the otolaryngologist. In a careful review of the case histories recorded by the authors cited in this publication we have not been able to find a single case except Novak's in which the pathologic manifestations were in the pharynx. This is peculiar in view of the relative abundance of lymphoid structure in this region and especially so when one considers that regions

which characteristically contain little lymphoid tissue may become the site of disease, as pointed out by Baehr.[7] We know of no good reason why this should be so.

The presence of markedly hypertrophied adenoids and tonsils in children is a matter of everyday routine observation with the otolaryngologist. The presence of adenoid tissue in the vault of the nasopharynx of an adult is not unusual. When, however, nasopharyngeal growths of lymphoid tissue assume tumor proportions in an adult, we cannot stress too strongly the necessity for immediate detailed clinicopathologic investigation.

Drs. F. E. Lejeune and Philip Bayon reviewed with us the case reported. The pathologic study was made by Dr. George H. Hauser. Dr. F. W. Gillig gave permission for the use of figure 1 and Dr. George H. Hauser for the use of figure 2.

165 Elk Place.

ALLERGY AS A CONTRIBUTING FACTOR TO BIOLOGIC DEAFNESS

CHUN-YUE MAO, M.D., M.Sc. (MED.)
PHILADELPHIA

Hereditary, or biologic, deafness in children is a matter of concern to laymen, otologists and educators alike. However, the general attitude is one of resignation to and acceptance of a problem which admits of no easy solution. Various investigators, notably Taylor [1] and Mosher,[2] have emphasized the importance of certain drugs as etiologic factors. Both of these authors implicated the drug quinine, the former on the basis of its known effect on uterine musculature, the latter on the basis of lesions observed in the cochleas of fetal guinea pigs. Maternal endocrine lacks have been discussed at great length. More recently vitamin deficiencies have been blamed for the development of hearing defects in the neonatal and early postnatal periods.

The present study was undertaken on the basis of recent comprehensive studies [3] which indicate clearly that allergy is definitely hereditary. Differences of opinion relate more to the immediacy of transmission than to the fact itself. Various adult diseases known to be allergic in character present symptoms due to acute edematous processes involving certain anatomic structures. Ménière's symptom complex, for instance, in certain cases responds favorably to the correction of proved allergic reactions in the labyrinth. These reactions are always related to some impairment of cochlear function.

It is known that in human beings only a single layer of cells separates the fetal from the maternal circulation. Might not, therefore, any sensitizing agents affecting the mother act directly on the undeveloped and undifferentiated cells of the fetal cochlea and the antibodies produced by the mother in response to sensitizing agents be transmitted directly

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From the Pennsylvania School for the Deaf.

Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Doctor of Medical Science (D.Sc. [Med.]) for graduate work in otolaryngology.

1. Taylor, H. M.: Prenatal Medication and Its Relation to the Foetal Ear, Surg., Gynec. & Obst. **64**:542 (Feb.) 1937.

2. Mosher, H. P.: Does Animal Experimentation Show Similar Changes in the Ear of Mother and Fetus After the Ingestion of Quinine by the Mother? Laryngoscope **48**:361 (June) 1938.

3. Duke, W. W.: Allergy, Asthma, Hay Fever, Urticaria and Allied Manifestations of Reaction, St. Louis, C. V. Mosby Company, 1925.

to the fetus? If this hypothesis is accepted, might not these sensitizing agents and antibodies, by repeated acts of such biologic trauma, weaken the auditory mechanism of the fetus and eventually contribute to the early failure of organs thus rendered inferior?

Studies by Hughson, Ciocco and Palmer [4] point out that the incidence of biologic impairment of hearing decreases rapidly with increasing age, from 1 to 3 years. Spain and Cooke [5] found that the larger the number of cases of hereditary sensitivity in a family the more marked the influence of heredity and the earlier the development of allergy in the children. Ciocco, Hughson and Palmer,[6] in a study of the auditory acuity of siblings, found 40 such related pairs in the Pennsylvania School for the Deaf. Examination of this group from the allergic point of view showed that only 4 of the pairs of siblings gave positive reactions—in all cases the deafness was of the biologic type. This 11 per cent incidence is not particularly striking in view of the general morbidity rates which follow.

The medical literature contains little information on tests for possible anaphylactic reactions on groups of supposedly normal children or on the incidence of protein sensitization in large numbers of normal and known pathologic subjects. Baker [7] found positive cutaneous reactions in 1.06 per cent of 937 normal children, and Peshkin and Rost,[8] in only 0.76 per cent of a series of 502 normal children. Brem and Colmes [9] reported that the total incidence of positive cutaneous reactions in 17 members of four control families was less than 1 per cent. Chown [10] stated roughly that 7 to 10 per cent of Canadians suffer from some allergic condition.

With this reported morbidity as a background, 535 resident pupils in the Pennsylvania School for the Deaf were tested for any allergic response to various sensitizing agents. In the performance of the allergic

4. Hughson, W.; Ciocco, A., and Palmer, C.: Studies of Pupils of the Pennsylvania School for the Deaf: I. Auditory Acuity, Arch. Otolaryng. **29**:403 (March) 1939.

5. Spain, W. C., and Cooke, R. A.: Studies in Specific Hypersensitiveness: XI. The Familial Occurrence of Hay Fever and Bronchial Asthma, J. Immunol. **9**:521 (Nov.) 1924.

6. Ciocco, A.; Hughson, W., and Palmer, C.: Studies on Pupils of the Pennsylvania School for the Deaf: II. Resemblances in the Auditory Acuity of Siblings, Human Biol. **11**:259 (May) 1939.

7. Baker, H. M.: Incidence of Protein Sensitization in the Normal Child, Am. J. Dis. Child. **21**:114 (Feb.) 1920.

8. Peshkin, M. M., and Rost, W. L.: The Incidence of Protein Sensitization in the Normal Child, Am. J. Dis. Child. **23**:51 (Jan.) 1922.

9. Brem, J., and Colmes, A.: Clinical and Potential Allergy in Families with Allergic Children, New England J. Med. **223**:329 (Aug. 29) 1940.

10. Chown, G.: Allergy in Children, Canad. M. A. J. **43**:141 (Aug.) 1940.

tests the scratch and the intracutaneous method were used. The procedures were simplified by the use of common allergens. The reading and recording were based on the size of the wheal, and the reactions were classified as +, ++, +++, and ++++, according to size. The scratch tests were made at least one to two weeks before the intracutaneous tests. The positive cutaneous reactions are presented in

TABLE 1.—*Incidence of Positive Allergic Reactions in Deafened Children*

Allergen	Reaction +	++	+++	++++	Total	Percentage
Intracutaneous Reactions in 153 Cases						
House dust	1	38	11	5	55	13.8
Spinach	..	27	3	4	34	8.8
Tomato	2	25	5	1	33	8.5
Crab	..	16	5	1	22	5.7
Cocoa	2	17	1	..	20	5.0
Asparagus	..	15	4	1	20	5.0
Coffee	2	13	2	..	17	4.3
Peanut	1	11	4	..	16	4.0
Orris root	..	9	3	2	14	3.5
Lima bean	1	12	..	..	13	3.3
Pineapple	1	11	1	..	13	3.3
Duck feathers	1	10	1	..	12	2.9
Malt	1	7	1	2	11	2.7
Codfish	1	7	1	..	9	2.2
String bean	..	9	..	..	9	2.2
Pork	1	6	2	..	9	2.2
Horse dander	1	8	..	..	9	2.2
Oyster	..	8	1	..	9	2.2
Yeast	2	6	1	..	9	2.2
Lettuce	1	6	..	..	7	1.7
Egg yolk	2	4	..	..	6	1.5
Carrot	2	4	..	..	6	1.5
Chicken	2	3	..	1	6	1.5
Pea	..	5	..	..	5	1.3
Rice	1	3	1	..	5	1.3
Rice powder	1	4	..	..	5	1.3
Sheep wool	..	5	..	..	5	1.3
Goose feathers	1	3	..	..	4	1.0
Egg white	3	1	..	..	4	1.0
Wheat	..	3	..	..	3	0.8
Orange	..	3	..	..	3	0.8
Beef	..	3	..	..	3	0.8
Milk	1	..	..	..	1	0.2
	31	302	47	17	397	100.0
Scratch Reactions in 17 Cases						
Timothy	4	9	2	3	17	54.8
Ragweed (giant)	..	5	1	3	9	29.0
Ragweed (dwarf)	1	..	2	2	5	16.2
	5	14	5	8	31	100.0

table 1. Nine allergens were used in the scratch tests on 535 children. Thus, a total of 4,815 scratch tests was obtained. In the intracutaneous tests thirty-three allergens were used, making a total of 17,655 intracutaneous tests.

The marked positive reactions to the inhaling allergens were correlated with the clinical findings. Eleven of 13 persons who gave ++++ cutaneous reactions to inhaling allergens had clinical manifestations. However, positive reactions to foods in these children were

not always associated with clinical manifestations. These findings are in fair agreement with the opinion expressed by Duke that cutaneous tests in cases of food sensitivity are often unsatisfactory.[3]

In table 2 it is to be noted that of 535 deafened children 283, or 53 per cent, were biologically deafened children, while 252, or 47 per cent, were pathologically deafened children. Of 283 biologically deafened children, 81, or 28.6 per cent, gave a positive reaction in the scratch or the intracutaneous test for allergy, while 72, or 28.5 per cent, of 252 pathologically deafened children gave a positive reaction. It suffices, therefore, to say, that the occurrence of allergy is by no means selective

TABLE 2.—*Allergic Reactions to Scratch and Intracutaneous Tests Among Five Hundred and Thirty-Five Pathologically and Biologically Deafened Children*

Biologically deafened children 283, or 53%		Pathologically deafened children 252, or 47%	
Positive reactions in 81, or 28.6%	Negative reactions in 202, or 71.4%	Positive reactions in 72, or 28.5%	Negative reactions in 180, or 71.5%

TABLE 3.—*Incidence of Positive Reactions in Deafened Children According to Age Groups*

	Biologically Deafened Children			Pathologically Deafened Children		
Age, Yr.	Sex	Cases	Positive Reactions	Sex	Cases	Positive Reactions
5 - 10	Male	30	2	Male	25	6
	Female	28	1	Female	17	2
10 - 15	Male	62	16	Male	58	12
	Female	51	19	Female	47	9
15 - 21	Male	66	30	Male	58	22
	Female	46	13	Female	47	21

so far as the two types of deafness are concerned but is definitely selective with regard to deafness and normal hearing.

Table 3 shows the age and sex incidence of the two groups of deafened children. Among 283 biologically deafened children the percentage incidence of positive reactions increased during the second decade. These findings do not agree with those of Spain and Cooke,[5] who reported a marked decrease in incidence after the first decade. There was no apparent preponderance for either sex. The same observations held true for pathologically deafened children.

SUMMARY AND CONCLUSIONS

From the foregoing data one fact is immediately apparent. In contrast to reported results on normal subjects, as previously mentioned, the total incidence of positive cutaneous reactions in the 535 seriously

deafened children examined was 28.5 per cent, while only 2 per cent showed definite clinical manifestations. This figure represents an incidence 27.44 per cent higher for the deafened children than for the largest numerical group of children with normal hearing reported. This greater incidence holds true irrespective of age or sex; in other words, it may be regarded as typical of this entire handicapped group.

Various facts present themselves as a result of the present study. Biologic deafness has been shown to develop with decreasing frequency from birth to 3 years of age. By biologic deafness is meant an intrauterine arrest or defect in the development of the auditory mechanism. This condition is present at or develops shortly after birth. Since this defect implies immaturity of cochlear structure, and hence inability to function, it may be considered final and irreversible. In the pathologically deafened children, with their equally high incidence of allergic manifestation, it may be assumed that the hereditary factor of allergy renders them constitutionally more susceptible to the influences of various contagious and infectious diseases. The evidence for these conclusions is undoubtedly more presumptive than specific. However, the percentages enumerated must be accepted as specific and definite under all conditions of examination, as outlined.

It is believed that definite evidence has been offered to indicate a higher percentage of allergic reactions in deafened children than in children with normal hearing. Allergy has been shown to be a hereditary disease in a high percentage of children so afflicted.

It is not unreasonable to assume that allergy and deafness in combination have some mutual hereditary background. If it were possible to make similar studies on the immediate forebears of these deafened children and on the children of their own generation and ancestry with normal hearing, one important factor in the development of hereditary deafness might well be solved.

7500 Germantown Avenue, Mt. Airy.

FUSOSPIROCHETAL ORGANISMS AND TONSILLITIS

BISMUTH THERAPY OF TONSILLITIS

JAMES FARRAR LEWIS, M.D.
FAYETTEVILLE, ARK.

Bacterial smears of mucopurulent material aspirated [1] from diseased tonsils frequently show considerable numbers of fusospirochetal organisms, while smears made from swabs of the same tonsils reveal only insignificant numbers of these organisms. What constitutes a significant number of these organisms is somewhat uncertain. The usual textbook picture of Vincent's angina calls for the presence of the organisms in predominant numbers in smears of material from tonsils presenting ulceration and membrane formation. I believe these diagnostic criteria are too exclusive, as I have encountered a great number of cases of follicular tonsillitis in which fusospirochetal organisms seemed to be the most important etiologic agent. Field,[2] likewise, observed that "milder [fusospirochetal] infections than those manifested by ulceration and membrane may occur." This author also commented that "grades of [fusospirochetal] infection in the throat and lungs, less severe than the usual textbook picture, are relatively frequent, and they are commonly not correctly diagnosed."

Vincent's organisms are present, along with various streptococci, staphylococci, pneumococci, etc., in the mouth and throat of almost all persons. They are fitted by nature to thrive under anaerobic conditions and are found in considerable numbers in gingival pockets which are the seat of chronic inflammation. They can be seen in large, even predominant, numbers in smears of aspirated material in many cases of chronic tonsillitis in children.[3] This suggests the hypothesis that these organisms produce chronic inflammation and hypertrophy in the tonsils. Thus, the Vincent organisms in the crypts may produce chronic inflam-

1. For aspiration I use a suction pump to which is attached a glass tube shaped like a medicine dropper, but longer. The wide end of the glass tube is pressed against the tonsil, and smears are made of the aspirated mucopurulent material (not necrotic plugs of tissue from crypts) which collects around the inside of the open end of the tube.

2. Field, H. Jr.: The Therapy of Fusospirochetosis, J. A. M. A. **114**:1073-1077 (March 23) 1940.

3. I have examined a few excised hypertrophied tonsils by means of the suction technic, without finding as many spirochetes as I expected. It may be that the ether given for anesthesia dried out the tissues and tended to disintegrate these organisms.

mation and swelling which closes off the mouths of the crypts, creating anaerobic pockets particularly favorable to the growth of these organisms, and so on in a vicious circle, leading to hypertrophy.

The degree of pathogenicity of Vincent's organisms has long been a controversial subject. That they can become invasive is proved by the fact that biopsy of typical ulcerative lesions shows these organisms penetrating deeply into apparently normal tissue, with the spirochetes leading the invasion, the bacilli next and various cocci in the outer zone.[4] The weight of experimental evidence and clinical experience favors the view that Vincent's organisms become invasive when a locus minoris resistentiae is presented by such factors as trauma, mercury or bismuth intoxication, malnutrition, abnormal dental conditions or primary invasion by some other organism.

Concurrent gingivitis and tonsillitis are seen with great regularity in children undergoing second dentition, when the element of trauma is at its highest. I have observed large numbers of Vincent's organisms in smears of material from the throat as well as from the gums of such children. In a recent paper [5] I described at some length cases of a similar coincidence.

Streptococci and Vincent's organisms are symbiotic.[6] Hemolytic streptococci are known to be present in most cases of acute tonsillitis, and they are generally thought to be the main etiologic agent.[7] My clinical observations, however, along with the study of smears (I have made no cultures), lead me to believe that fusiform bacilli and spirochetes play an important role in acute, as well as in chronic, tonsillitis.

I encountered cases of acute tonsillitis with more or less evidence of active fusospirochetal infection frequently during the winters of 1938-1939 and 1939-1940. Not many were encountered this past winter (1940-1941), which was marked rather by frequent pneumococcic nasopharyngeal infections, without much follicular tonsillitis and without much evidence of activity of the fusospirochetal organisms in the tonsils. A few selected cases of more or less chronic fusospirochetal tonsillitis are now presented.

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5. Lewis, J. F.: Vincent's Angina and Tonsillitis: Bismuth Treatment, J. Arkansas M. Soc. **38**:39-44 (July) 1941.

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7. Dawson, M. H., in Cecil, R. L.: A Textbook of Medicine, ed. 5, Philadelphia, W. B. Saunders Company, 1941.

REPORT OF CASES

CASE 1.—T. L., a boy aged 7 years, had chronic tonsillitis with marked hypertrophy of several years' standing. During the winter of 1938-1939 he had frequent attacks of acute follicular tonsillitis, with temperatures ranging from 101 to 104 F. During this time the gums were often sore from loosening of baby teeth and eruption of permanent teeth. There was no ulceration, but smears of material from the gums showed many Vincent's organisms (3 plus). I found that smears of material aspirated from the tonsils showed considerable numbers (2 to 3 plus) of fusospirochetal organisms during the acute attacks and moderate numbers (2 plus) between attacks. (Unfortunately, there is no way to express the degree of positivity of smears for Vincent's organisms; for my own notes I usually grade the smears 1 to 4 plus.)

The acute attacks could be terminated with remarkable promptness, so that the patient was able to return to school in a day or two, by intravenous administration of a small dose of mapharsen. Before I began this therapy the patient had been going through prolonged bouts of tonsillitis, without showing membrane formation typical of Vincent's angina, however. Later I found that an intramuscular injection of a bismuth compound would work about as well as the arsenical, though not quite so promptly, perhaps.

As soon as school was out the patient's tonsils were removed, and he was well (gaining 15 pounds [6.8 Kg.]) all through the following winter.

CASE 2.—H. M., a girl aged 18, came to the office with the complaint of constant bad breath. (I observed a distinct fetor.) She gave a history of having "trench mouth" at the age of 9 years. Her teeth and gums appeared normal, and the smears revealed only rare Vincent's organisms in material from between the teeth, but smears of material aspirated from the tonsils showed myriads of the organisms. The tonsils showed pronounced hypertrophy. The patient did not have sore throat at this time and had not had one for a number of months but had had several attacks during the previous year. An injection of a bismuth compound was given on Jan. 23, 1939. On January 28 the tonsils were observed to be smaller and the oral fetor was gone. The second injection of the bismuth preparation was given. On February 13 the tonsils were observed to be only one-third their former size. Two more injections of the bismuth compound were given, but smears of material aspirated from the tonsils continued to show a moderate number (2 plus) to "myriads" of Vincent organisms. Three injections of mapharsen were given, but no further change in the picture was obtained. The tonsils later assumed their former size, and the oral fetor also returned. Tonsillectomy was performed later.

CASE 3.—L. S., a girl aged 16, had chronic tonsillitis, with great hypertrophy of the tonsils, and chronic gingivitis. Because of the presence of many fusospirochetal organisms in smears of material from the tonsils I decided to try "specific" treatment. The patient was given three injections of neoarsphenamine at intervals of seven days or so. On March 20, 1941 the right tonsil was reduced to one-half the usual size. Later the tonsils returned to their previous dimensions, and tonsillectomy was performed.

CASE 4.—A. M. E., a woman aged 21, was treated in April 1939 for severe tonsillitis. Smears from swabs of material from the tonsils showed very few fusospirochetal organisms. Gargles of hot saline solution, local application of a silver nitrate solution and sprays of a solution of sodium perborate were used, but the

patient had high fever for three days. Convalescence was slow. Severe urticaria developed on the fourth day, probably from bacterial allergy. The patient complained of tenderness of her gums at this time, and smears showed the presence (2 plus) of Vincent organisms. There was some inflammation of the gums but no ulceration.

Over a year later the patient had an attack of severe tonsillitis, with inflammation and swelling but no exudate. The temperature was 103 F. I made a smear from a throat swab and proceeded with an injection of a bismuth preparation. The smear, studied later, showed occasional spirochetes and fusiform bacilli. The patient was practically well in twenty-four hours. Hives developed after this illness, as in the previous attack, suggesting a similar etiologic agent.

Case 5.—D. G., a boy aged 2½ years, had tonsillitis in July 1938, with low grade fever for many days. A cough lingered for about a month. The patient had tonsillitis again in February 1939, followed by otitis media. In spite of paracentesis of the drums and treatment with sulfanilamide and azosulfamide, mastoiditis developed, necessitating mastoidectomy. The etiologic agent was not determined. Repeated smears made from throat swabs had not shown evidence of active fusospirochetal infection.

In May 1939 tonsillitis again developed, and I made smears of material obtained by the suction technic, finding a great many long slender bacilli, a few of which showed some curving. These may have been atypical fusospirochetal organisms. Whether these organisms undergo pleomorphism is controversial.

An injection of sulfarsphenamine was given, and the patient was well in forty-eight hours. This prompt recovery was striking in view of previous experience with this patient. What his subsequent course has been I do not know, as the family moved to another state shortly thereafter.

Case 6.—S. Y., a girl aged 16 months, whose case was somewhat similar to that of D. G.'s, was treated in November 1939 for tonsillitis followed by an unusually troublesome cough. Smears from throat swabs showed very few Vincent's organisms. The cough persisted, and finally, five weeks after onset of the tonsillitis, a smear made of material obtained by the aspiration technic showed large numbers of Vincent's organisms. The patient was given an injection of a bismuth compound, and thirty-six hours later her mother reported that the effect was "almost miraculous"—the cough had disappeared. Some days later the cough returned, and a second injection of the bismuth preparation was given, with prompt benefit.

The patient probably had fusospirochetosis of the lower respiratory tract, as well as a tonsillar infection.

Case 7.—Mrs. C. C. G., aged 65, was treated on Feb. 21, 1939 for tonsillitis of ten days' duration, associated with a foul breath. The patient was edentulous. Smears showed moderate numbers of Vincent's organisms. An injection of a bismuth compound was followed by surprisingly prompt recovery. A year later I observed that the tonsils were still huge and harbored many spirochetes and fusiform bacilli, both types of organisms appearing unusually small. The patient had had no recurrence of sore throat.

Case 8.—G. C. S., a boy aged 7, was treated on Jan. 7, 1939 for Vincent's angina with membrane. Smears obtained from a swab of the throat showed typical fusospirochetal organisms. The patient had gingivitis associated with second dentition. There was a history of frequent tonsillitis. His sister, aged 10, also had frequent

attacks of tonsillitis. The father had severe pyorrhea. The patient was able to return to school a few days after being given two injections of a bismuth compound. He had several attacks of tonsillitis that spring, and I was able to cut these attacks short each time with a single injection of a bismuth compound.

I did not see the patient at all the next winter, but on June 25, 1940 he came to my office with a sore finger and I observed that his teeth and gums were healthy and his tonsils only about half as large as they were a year before. He told me he had not had a sore throat in a number of months. On July 9, 1940, however, I was called to see him for severe follicular tonsillitis (temperature, 103 F.) and gave him an injection of a bismuth compound. The next day his father reported that the boy was almost well.

CASE 9.—D. M., a man aged 22, has been under my observation for three years, during which time he has had many attacks of follicular tonsillitis (with smears giving evidence of fusospirochetal origin) and two or three attacks of typical Vincent's angina. He had chronic (fusospirochetal) gingivitis associated with decay of almost all his teeth along the gum line. I advised him to have gold fillings put in, as the commonly used silver fillings are likely to cause irritation and infection where they are in contact with the gums, but he was unable to have this done. He finally decided to have all the teeth extracted but has been dilatory in attending to this. The frequent gingival and tonsillar infections of this patient have always responded promptly to injections of bismuth.[7a]

Obviously, medical therapy is not likely to have any permanent effect on tonsils which are already greatly hypertrophied. If it is true, as I suspect, that Vincent's infection plays a large part in the development of chronic tonsillitis, then the most important indication is prevention by careful dental and medical observation and treatment of concurrent gum and throat infections during the periods when the child's gums are irritated, traumatized and infected—particularly during dentition. Valuable medicaments for local and systemic use in combating Vincent's infection are now available. Arsenicals and bismuth are widely used. Nicotinic acid may prove effective.[8] Systemic treatment of Vincent's infections with sulfanilamide and its derivatives has not been found particularly effective, but local application of sulfanilamide powder may prove helpful in gingival infections.

Bismuth has long been recognized as a potent agent in treatment of Vincent's infection. Harrell,[9] in 1931, after making a general review of the literature on Vincent's angina, stated that "the use of bismuth preparations constitutes the best form of treatment, for they are less toxic, more powerful and more economical." I believe the reason bismuth is not

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7a. This patient has not had an attack of tonsillitis in the eight months which have now elapsed since his teeth were extracted.

8. King, J. D.: Vincent's Disease Treated with Nicotinic Acid, Lancet **2**: 32-35 (July 15) 1940.

9. Harrell, V.: The Present Status of Plaut-Vincent's Infection, Arch. Otolaryng. **14**:1-8 (July) 1931.

more generally used for Vincent's angina is that few physicians realize the limitations of systemic medical therapy alone for *gingival* infections. They are likely to expect a cure from injections or local applications alone, and failing to obtain consistent cures from one drug, they change to others, and so on. Local dental care is of the utmost importance in achieving cure and preventing relapse. This includes eradication of anaerobic pockets about teeth, correction of dental abnormalities (such as accumulations of tartar, malocclusion, etc.), proper use of the toothbrush and perhaps the employment of hot saline mouth washes. The mouth washes are often surprisingly effective.[9a]

In the six months during which I studied the cases presented I treated about 40 patients with acute tonsillitis who showed enough evidence of tonsillar fusospirochetal infection to justify the use of "specific" treatment. Either arsenicals or bismuth injections usually produced prompt improvement. I came to feel that use of bismuth was the treatment of choice in perhaps the majority of cases of severe tonsillitis. When bismuth is used in selected cases of acute tonsillitis, the temperature usually becomes normal within twenty-four hours and there is marked subjective relief within twelve hours. Exudate and swelling show considerable clearing within twenty-four hours.

On making a review of the literature, I found that Monteiro [10] and many of his colleagues in Brazil had been using bismuth for several years in the treatment of acute nonspecific tonsillitis, as well as of Vincent's angina. They have made extensive bacteriologic investigations, and they, like workers in this country, regard acute tonsillitis as primarily a streptococcic infection. There are many enthusiastic reports in foreign journals [11] confirming the efficacy of this treatment.

Whether its effectiveness is due to streptococcicidal or to fusospirocheticidal activity or to both, bismuth is a highly effective agent in the treatment of most types of acute tonsillitis. I have found it ineffective in the management of pneumococcic nasopharyngitis, a point that

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9a. Dorland, C. K.: Personal communication to the author.

10. Monteiro, A.: Une nouvelle thérapeutique. Le bismuth dans le traitement des angines aiguës nonspécifiques, Ann. d'oto-laryng., June 1934, pp. 577-566; Estado atual da bismutoterapia nas anginas aqudas, Rev. brasil. de oto-rino-laring. **7**:121-157 (March-April) 1939.

11. Boese, O.: Behandlung der akuten eitrigen Angina mit Wismutsalzen, Deutsche med. Wchnschr. **62**:776 (May 8) 1936. Buhre, G.: Die Behandlung der Angina mit Wismutinjektionen, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:307, 1939. Gipperich, L.: Nuovo trattamiento dell'angina cal preparati di bismuto, Gior. di clin. med. **18**:531-535 (June 10) 1937. Mayersohn, L.: Nouveau traitement des angines aiguës simples, Folia oto-laryng. orient. **3**:52-56, 1936. Stiehr: Erfahrungen bei der Behandlung eitrigen Anginen mit einem Wismutpräparat (Etosio), München. med. Wchnschr. **44**:1761 (Nov. 1) 1935. Galewski, S.: Treatment of Tonsillitis with Bismuth, Lancet **2**:636 (Nov. 16) 1940.

Monteiro has likewise stressed. I have generally used the standard bismuth subsalicylate in oil, as it is promptly effective, though slowly absorbed, and rarely causes pain, while the soluble bismuth preparations frequently do cause pain afterward. Needless to say, all proper precautions are taken to avoid injecting the oil into a blood vessel. A single injection of 1 cc. of the subsalicylate (containing 0.13 Gm. of the drug, equivalent to 76 mg. of metallic bismuth) is usually adequate for an adult with acute tonsillitis. In treating children I have used doses such as are employed in treating congenital syphilis (10 mg. of metallic bismuth for infants; 20 to 50 mg. for children). Monteiro and his colleagues found much smaller doses adequate, I believe, employing soluble preparations. Monteiro [12] stated that he generally used 1 to 2 mg. for infants, 2 to 4 mg. for children between 2 and 8 years of age, 5 mg. for children between 8 and 12 years of age and 10 mg. for persons above the age of 15. The commonest toxic effect of bismuth is stomatitis, with secondary fusospirochetal infection, but stomatitis develops only on the basis of prolonged antisyphilitic treatment. I have seen only 3 cases of tonsillitis in which toxic effects resulted from this treatment. Two infants showed cutaneous eruptions, which may have been due to the use of a rather large dose in each case. (According to Moore,[13] such eruptions contraindicate futher administration of the drug.) I have seen a child (not my patient) with temporary anuria following the administration of 3 ampules of a soluble bismuth preparation (containing 76 mg. of metallic bismuth per ampule) in three days.

It may be that an oral bismuth preparation will prove useful in the treatment of tonsillitis, obviating the necessity of an injection.

In the treatment of tonsillitis I have used bismuth in preference to sulfanilamide because it has been my impression that it is more effective, though I have not compared the effects of the two drugs in any series of cases. It is certainly less toxic. Long and Bliss,[14] in discussing sulfanilamide therapy of acute streptococcic tonsillitis and pharyngitis, remarked ". . . this disease does not lend itself to statistical analysis, and the observer has to depend upon subjective impressions which may lead him astray." The same is obviously true of bismuth therapy of acute tonsillitis. Long and Bliss reported that it seemed to them that sulfanilamide was effective in the treatment of streptococcic infecions of the tonsils and pharynx, and many other observers have reported like-

12. Monteiro, A.: Personal communication to the author.

13. Moore, J. E.: The Modern Treatment of Syphilis, Springfield, Ill., Charles C. Thomas, Publisher, 1933.

14. Long, P. H., and Bliss, E. A.: The Clinical and Experimental Use of Sulfanilamide and Sulfapyridine and Allied Compounds, New York, The Macmillan Company, 1939.

wise; but Rhoads and Afremow,[15] among others, reported unfavorably on the use of sulfanilamide in the treatment of sore throat due to hemolytic streptococci.

SUMMARY AND CONCLUSIONS

Several selected cases of acute and chronic tonsillitis are presented in which the results of laboratory examination, the clinical course and the response to arsenical and bismuth therapy indicated that the Vincent organisms were important etiologic factors.

Vincent's organisms (with other bacteria) are present in greater or less numbers in most cases of tonsillitis, and they are probably more active than is generally supposed.

Bismuth has been found to be highly effective in the treatment of tonsillitis and is recommended in cases of the severe form. Bismuth is not effective in treatment of pneumococcic nasopharyngitis.

Lewis Building.

15. Rhoads, P. S., and Afremow, M. L.: Sulfanilamide in the Treatment of Sore Throat Due to Hemolytic Streptococci, with Controls, J. A. M. A. **114**:942-943 (March 16) 1940.

OCCUPATIONAL DEAFNESS: AUDIOMETRIC OBSERVATIONS ON AURAL FATIGUE AND RECOVERY

A PRELIMINARY REPORT

DOUGLAS CHAMBERLAIN, M.D.
CHATTANOOGA, TENN.

Perhaps only by repeated audiograms made before, during, immediately after and some time after various separate harmful influences are at work can one study completely the respective effects on the hearing. Such a method would seem to be more satisfactory than one which depends on audiograms made after harm has been done. The latter method offers no way of distinguishing between the effects of the many factors at work over a period of time; whereas, if each possible influence can be studied separately, one may hope eventually to appreciate the damage done by each. How, for example, does one distinguish between the fatigue or damage from exposure to noise and the toxic effects of quinine and other drugs? According to most writers, these factors produce much the same effect on the hearing as shown in a final audiogram, but how large a part does each of any number of other possible noxious influences play? What factors make the inner ears of one man more susceptible to damage from noise or drugs than those of another? Does each exposure to loud noise produce in addition to aural fatigue small but definite cochlear damage, with an accumulative effect after many exposures? Or may complete recovery be accomplished after each trauma and permanent cochlear damage supervene only if the trauma is repeated before recovery is complete?

The admirable papers by Bunch [1] and by Larsen [2] are based largely on studies of audiograms and results of hearing tests made sometimes after months or years of exposure to occupational noise. The loss of hearing which they reported is definite and in most cases permanent. Other factors which may have been at work during the period of

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The management and employees of the Chattanooga Boiler and Tank Company cooperated in making these observations possible.

1. Bunch, C. C.: The Diagnosis of Occupational or Traumatic Deafness: A Historical and Audiometric Study, Laryngoscope **47**:615 (Sept.) 1937.

2. Larsen, B.: Investigations of Professional Deafness in Shipyard and Machine Factory Laborers, Acta oto-laryng., 1939, supp. 36.

exposure are not known. Larsen's subjects were examined in the evening, after work, but it is not known at what period of their recovery from the day's aural fatigue the tests were made. Fifteen of his subjects, all laborers, were retested after periods of unemployment, and about 25 per cent of these showed some improvement. It may be, however, that this 25 per cent would have shown the improvement within a few hours of the original test. On the other hand, the 75 per cent whose hearing was unimproved may have recovered from the aural fatigue of the day's work by the time the original tests were made. He subjected two of his friends and himself, all about 35 years of age, to the noise inside a boiler from one-half to one hour while riveters were at work. All three showed a loss of from 30 to 45 decibels in the region of 4096 double vibrations, which later completely disappeared.

Dickson, Ewing and Littler [3] reported the audiograms of one of the authors and a pilot made within ten minutes after a flight in a twin-engine bomber in which there was no attempt to protect the ears. Both audiograms showed a characteristic dip of from 30 to 45 decibels at 4096 double vibrations and 8192 double vibrations, with no appreciable loss for other frequencies. Recovery followed in a few hours.

Campbell and Hargreaves [4] reported audiograms of one of the authors showing the effects of exposure to airplane noise. The experiments consisted of six one-hour periods of flight on different days, six two-hour periods of flight and four four-hour periods of flight. The loss occurring after the four-hour periods of flight was the greatest and amounted to 25 decibels for 4096 double vibrations, with a widening of the gap to extend from 1024 to 8192 double vibrations. They observed that complete recovery in each of these experiments followed a period approaching the square of the time during which the fatiguing force was applied and expressed the thought that this fitted into the general physiologic concept of fatigue. These observations are important in illustrating complete aural recovery as shown by the audiometer if the rest period is adequate. They expressed the belief, however, that constant repetition of the insult had an accumulative effect resulting in permanent loss of hearing.

Audiograms of boilermakers exposed to the noise and vibrations of their daily occupation made immediately before, immediately after and at intervals after such exposure have not been found in the literature. Such audiograms might confirm the observations of Campbell and Hargreaves on the length of the period of recovery from aural fatigue

3. Dickson, E. D. D.; Ewing, A. W. G., and Littler, T. S.: The Effects of Aeroplane Noise on the Auditory Acuity of Aviators, J. Laryng. & Otol. **54**:531 (Sept.) 1939.

4. Campbell, P. A., and Hargreaves, J.: Aviation Deafness—Acute and Chronic, Arch. Otolaryng. **32**:417 (Sept.) 1940.

and might show that permanent damage to the hearing may be delayed or avoided by the simple expedient of permitting recovery to continue over an adequate period after a day's exposure to excessively loud noise.

REPORT OF CASES

CASE 1.—J. W., a white man aged 32, at the time of examination was a boilermaker, employed at chipping and caulking with a small pneumatic hammer. He had worked in a boiler shop sixteen years. He had done no steel riveting himself for about six years. As a child he had an abscessed right ear but had not suffered from otitis media since. Some "escaping steam" tinnitus had been noted for ten to twelve years, worse in the right ear and with colds. Quinine taken for "malaria" five years previously caused some temporary deafness and tinnitus. He

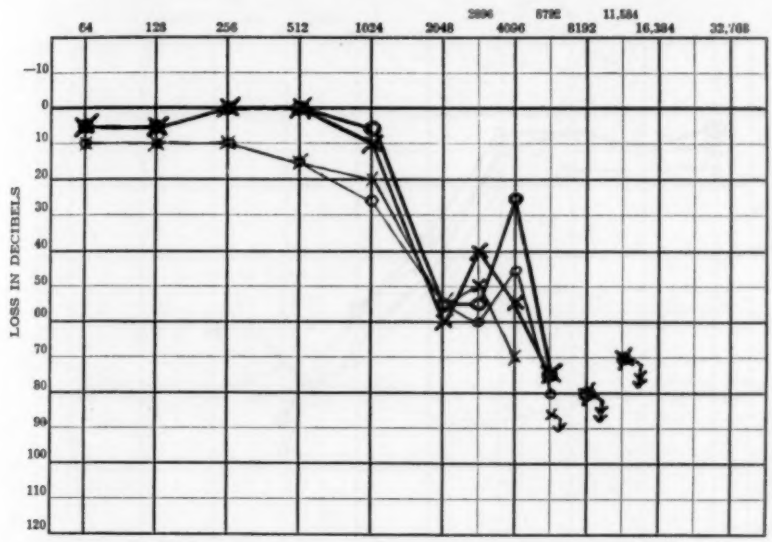

Chart 1 (case 1).—Circles represent the right ear; crosses the left ear. The heavy lines were recorded early in the morning, before work; the light lines, after the day's work caulking a 3 ft. steel pipe in the open. See the text for a complete description of the conditions.

stated that he smoked ten cigarets a day. Several brothers are boilermakers and are slightly deaf.

Otoscopic Examination.—A healed perforation was seen in the right drumhead with a calcium deposit in the anterior quadrant. Otherwise the drumheads were normal.

Audiogram.—The *heavy lines* were recorded Sept. 17, 1940, early in the morning, before work. The *light lines* were added immediately after the caulking of a 3 foot (91.5 cm.) steel pipe in the open from 8 a. m. to 3:30 p. m. with one-half hour off for lunch. The left ear was nearest the work. Roaring tinnitus was experienced after work. Early in the morning of March 12, 1941, at the same place, the audiogram was essentially the same as on the morning of Sept. 17, 1940 (lines not shown). On March 12, 1941, the Weber test showed reference of sound to the left

ear, and the Rinne test with the 512 double vibration steel fork was positive for both ears.

Result.—Slight temporary loss from fatigue was present for frequencies from 64 through 1024. No significant further loss was observed after six months, although the subject had done work of the same sort regularly three to four days a week.

Case 2.—C. S., a white man aged 43, had been employed as a boilermaker for twenty-five years. He considered his hearing good for his age. In 1925 welding largely replaced the old hammer riveting, a change which greatly reduced the noise to which he had been exposed prior to that year. He used no ear protection. Tinnitus had not been noted except after taking a capsule for "flu," in 1918. There had been no ear infections. Quinine had not been taken. His father was a boilermaker at 72 and a little deaf.

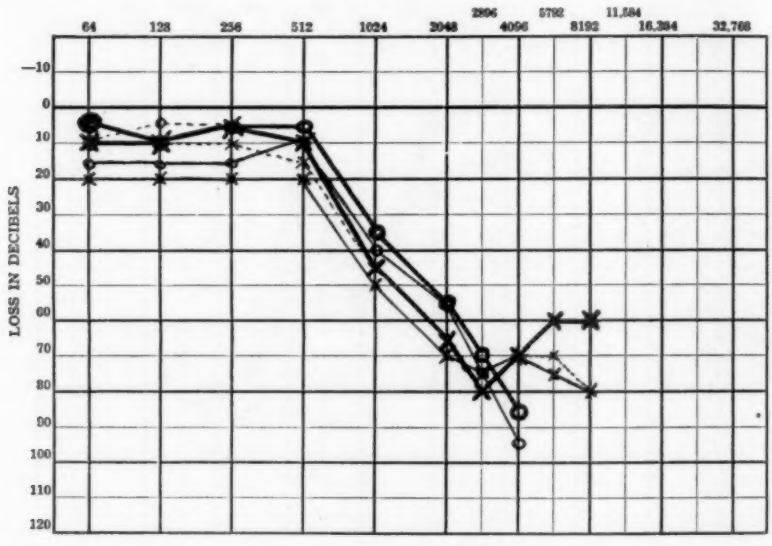

Chart 2 (case 2).—Circles represent the right and crosses the left ear. The heavy lines were recorded before work; the light lines, ten minutes after the day's work at riveting; broken lines, after a night's rest (fifteen hours). See the text for a complete description of the conditions.

Otoscopic Examination.—The findings were normal.

Audiogram.—The *heavy lines* were recorded at 7 a. m. Monday, Sept, 16, 1940, before work, after forty-four hours of complete rest from noise. The *light lines* show the hearing ten minutes after he stopped work at 3:30 p. m.; a half hour had been allowed for lunch. The worker used a large pneumatic riveter outside a boiler 11 ft. (3.5 meters) in diameter by 19 ft. (5 meters) long in an enclosed shed. The left ear was nearer the work than the right ear. The *broken lines* show complete recovery within fifteen hours, the following morning, except as shown by these lines.

Result.—There was a marked permanent loss, probably of long standing. Slight additional loss was caused by noise. Essentially complete recovery to the level before exposure to noise occurred within fifteen hours.

CASE 3.—J. K., a white man aged 32, had been employed as a boilermaker for five weeks at the time of examination and for six months eleven years previously. He described his occupation as "bucking rivets" within boilers. He also had done odd jobs. He had not used pneumatic hammers. He considered his hearing good.

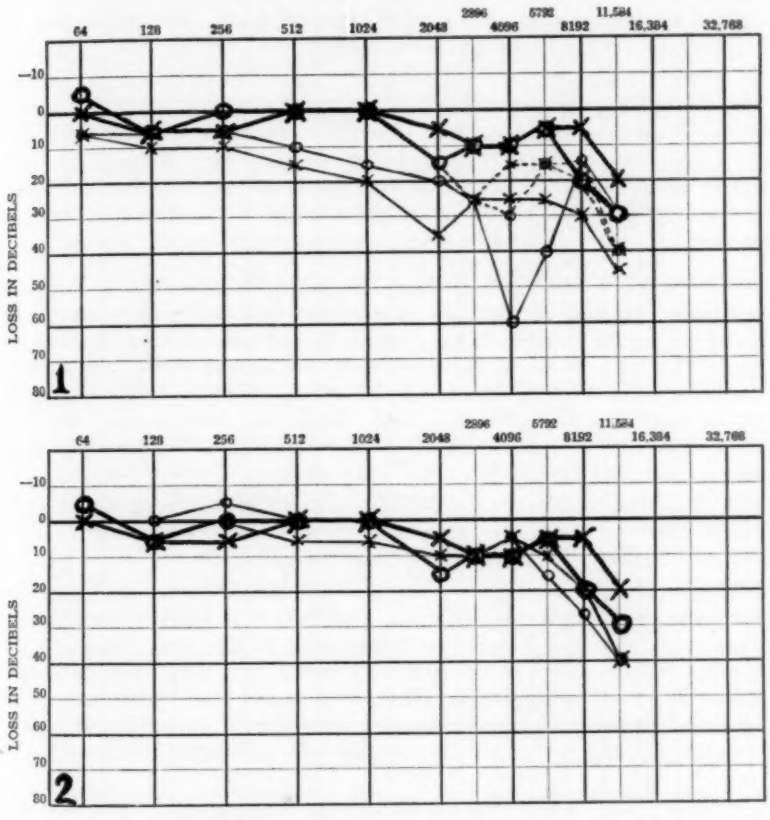

Chart 3 (case 3).—Circles represent the right and crosses the left ear. In audiogram 1, the heavy lines were recorded before work, the light lines forty minutes after the day's work at "bucking rivets" inside the boiler on which the worker in case 2 was working; the broken lines were added after a night's rest (fifteen hours). In audiogram 2, the heavy lines are the same as in audiogram 1; the light lines were added six months later, with no exposure to similar excessive noise in the interim. See the text for a complete description of the conditions.

He mentioned frequent head colds and had a slight cold at examination. No ear infections were recalled. There had been occasional momentary high-pitched tinnitus. He used no ear protection while working. He always took a little quinine for colds. There had been no ringing except once, a year ago, with quinine. He smoked three sacks of tobacco a week. The family history was without bearing.

Otoscopic Examination.—The results were negative.

Audiogram 1.—The *heavy lines* were recorded early Monday morning, Sept. 16, 1940, before the patient went to work, after forty-four hours of complete rest from noise; the *light lines*, forty minutes after he had stopped working from 8 a. m. to 3:30 p. m., with a half hour off for lunch, "bucking rivets" inside the boiler on which the worker in case 2 was employed. The left ear was nearest the rivet. The *broken lines*, obtained the next morning, after fifteen hours' rest, reveal that recovery was complete except as shown by these lines.

Audiogram 2.—The *heavy lines* are the same as in audiogram 1. The *light lines* were added six months later at the same time and place. The patient had not been exposed to similar excessively loud noise in the interim. The Weber test showed sound referred to the right; the Rinne test was positive for both ears with the 512 double vibration steel fork.

Result.—There was significant loss after noise for all tones above 256 double vibrations except for 8192 double vibrations in the right ear. Recovery for tones above 2048 double vibrations had not taken place in fifteen hours, but six months later the hearing was back to the starting point except for slight loss at the three highest tones.

Case 4.—G. C., a white man aged 32, was employed at handing up hot rivets within a boiler. He also did odd jobs. He had not used pneumatic hammers. He considered his hearing normal. At examination he had been working three months, and worked for three weeks in December 1939. He noticed considerable deafness for two hours after working inside a boiler during riveting. Outside work did not bother him. No protection was used. There had been no ear infections. Infections of the upper respiratory tract were infrequent. He experienced ringing tinnitus for thirty minutes to two hours after working. Quinine had been taken for only one week eleven years previously. No tinnitus was noted then. He smoked 20 cigarets a week. The family history was without bearing.

Otoscopic Examination.—The findings were not important.

Audiogram 1.—The *heavy lines* were recorded early Monday morning, Sept. 16, 1940, before the patient went to work, after forty-four hours of complete rest from noise; the *light lines*, twenty-five minutes after all day work handing up hot rivets inside the same boiler as the worker in case 3, with a half hour for lunch; the *broken lines*, the next morning, after fifteen hours' rest. Between the light lines and the broken lines might be sketched other lines representing hearing loss after exactly the same work the following day with petrolatum-covered cotton in both ears.

Audiogram 2.—The *heavy lines* are the same as in audiogram 1. The *light lines* were added six months later, with no exposure to similar excessively loud noise in the interim. The Weber test showed sound referred to the right; the Rinne test was positive for both ears with the 512 double vibration steel fork.

Result.—There was marked loss for all except the three lowest tones after loud noise. A considerably greater loss was shown by this worker than by the previous worker, although they had endured the same noise the same length of time. It is doubtful if being tested fifteen minutes nearer the cessation of the noise could explain this difference. Recovery was by no means complete in fifteen hours. At the end of six months the left ear had not recovered the hearing for tones above 1024 double vibrations. The right ear, on the other hand, had not only recovered but had shown a definite improvement for practically all tones except 8192 double

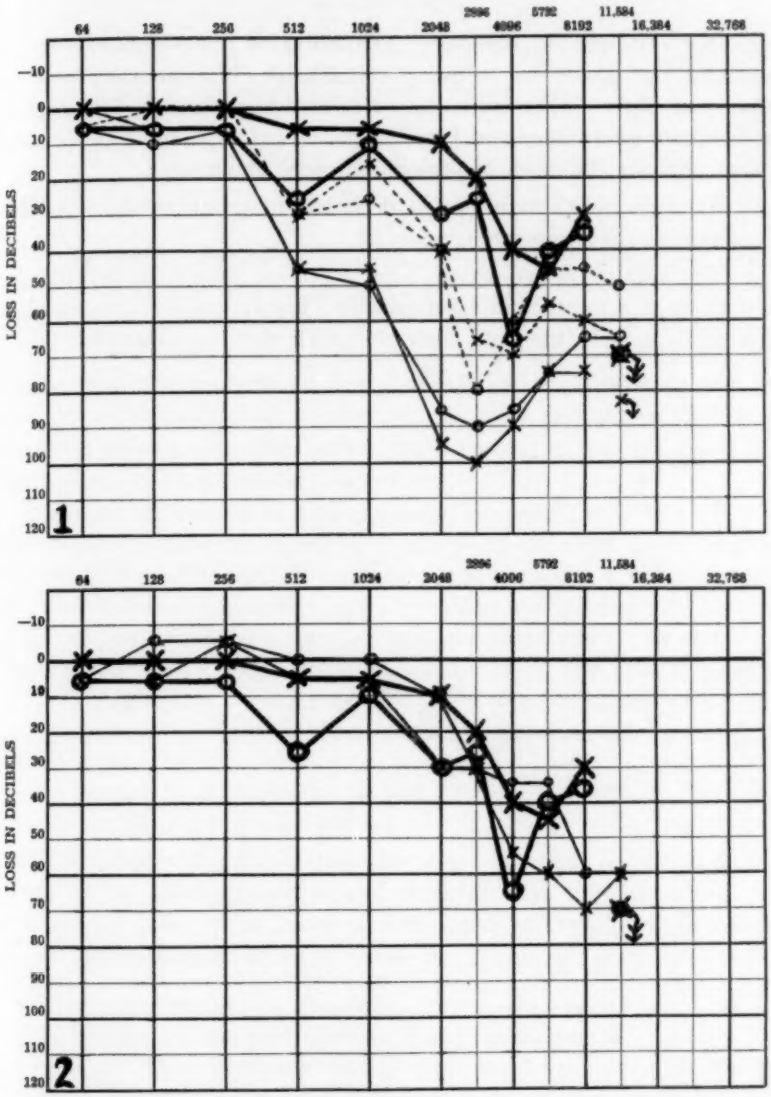

Chart 4 (case 4).—Circles represent the right and crosses the left ear. In audiogram 1, the heavy lines were recorded before work, the light lines twenty-five minutes after the day's work handing up hot rivets inside the same boiler as the worker in case 3; the broken lines were added after a night's rest (fifteen hours). In audiogram 2, the heavy lines are the same as in audiogram 1. The light lines were added six months later, with no exposure to similar excessively loud noise in the interim. See the text for a complete description of the conditions.

vibrations, suggesting that the original Monday morning audiogram did not represent complete recovery from similar noise on the preceding Saturday, forty-four hours before.

CONCLUSIONS

1. Aural fatigue in the daily occupation of a boilermaker may be great and the power of recovery remarkable, but recovery does not always take place within fifteen hours of rest from noise. This suggests that adequate intervals of rest between periods of exposure to noise might prevent or delay the onset of permanent cochlear damage.

2. The ears of one man may be definitely more susceptible than those of another to the same fatiguing noise.

3. The high tones are usually affected much more than the low ones, but if there is a permanent high tone loss before exposure to noise, the lower tones may be the only ones for which there is significant fatigue.

CONGENITAL ATRESIA OF THE POSTERIOR NARES

REPORT OF TWO CASES

ALFRED A. SCHWARTZ, M.D.
AND
HAROLD J. ISAACS, M.D.
NEW YORK

The condition known as congenital atresia of the posterior naris is caused by a vertical wall of tissue lying in a plane perpendicular to that of the septum and situated between the posterior ends of the turbinates and the eustachian orifice. In cases of complete atresia this wall merges with the margins of the choanal orifice, separating the naris from the nasopharynx. It is concave anteriorly and bulges slightly into the nasopharynx. In the upper central portion one or more small indentations or foramens may be present.

The earliest record of this anomaly, according to Otto,[1] is to be found in an article by Roederer,[2] published in Göttingen, Germany, prior to 1755. Otto cited this article as the authority for this statement that "sometimes the posterior nasal openings are lacking, due to a congenital anomaly." In a later edition of the same work [3] he stated: "In congenital closure of the hinder nasal openings, the palate bones are very much deformed." Later in 1841 he [4] reported 3 cases of its occurrence in monsters. One of these monsters was human; the other two were animals.

In a survey of the literature in 1940, Cinelli [5] stated that the number of cases reported was about 220.

It is universally considered that the condition is congenital. In regard to the pathogenesis, several theories have been proposed. The prevailing opinion of the authors of the most recently published articles is that the condition develops from the failure of obliteration of the

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From the laboratory of the Department of Otolaryngology, Montefiore Hospital.
This investigation was made possible through a grant from Mr. Arthur O. Dietz.

1. Otto, A. W.: Handbuch der pathologischen Anatomie des Menschen und der Thiere, Breslau, J. F. Barth, 1814, p. 203.

2. Roederer, J. G.: Descriptio foetus parasitici; cited by Otto.[1]

3. Otto, A. W.: Lehrbuch der pathologischen Anatomie des Menschen und der Thiere, Berlin, A. Rücker, 1830, p. 185.

4. Otto, A. W.: Monstrorum sexcentorum descriptio anatomica, Breslau, F. Hirt, 1841, cases 101, 106 and 554.

5. Cinelli, A. A.: Nasal Atresias: A Surgical Critique, Ann. Otol., Rhin. & Laryng. **49**:912 (Dec.) 1940.

bucconasal membrane of Hochstetter [6] (Schaeffer,[7] Morgenstern,[8] Donnelly,[9] Anderson [10]). Fraser [11] also accepted this view. Lang,[12] in addition, suggested that the condition may be hereditary and cited the case of a boy of 15, his sister, 16, and their mother, 35, all with complete bony atresia on the right side. Another child, who died at the age of 5 weeks, had similar obstructive symptoms on the right side. A maternal aunt of the mother, dead at the time, also had had symptoms pointing to atresia on the right side.

The malformation may be unilateral or bilateral, the unilateral type being the more frequent. It may be partial or complete. Partial atresia is rarely seen or recognized, for it causes no obstruction and hence gives no indication of its presence.

The structure of the occluding wall may be membranous or bony or part membranous and part bony. Cartilage may also be present. The thickness of the partition varies greatly. If the wall is membranous, it is usually thin. If bony, it may vary in thickness from a few millimeters to 12 millimeters, and possibly more. It is thickest below, at the palate and septum, and gradually thins out laterally and toward the roof of the nose.

The cardinal symptom of congenital atresia is the inability to breathe through the affected nostril. A unilateral obstruction in infancy usually gives little indication of its presence. There may be some difficulty in breathing and some interference with the taking of nourishment. It seems, however, that if sufficient air can pass through one nostril the infant's general health is not seriously impaired. A tenacious discharge is usually observed dripping from the affected nostril.

In newborn infants an additional factor must be taken into consideration. Although there is as yet little proof that bilateral choanal atresia is responsible for all or even the greater part of the deaths in cases of asphyxia neonatorum, there is sufficient evidence to show that it has been the cause of death in several instances. In a case reported by Ronaldson,[13] in which there was a bilateral membranous occlusion, the

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6. Hochstetter, F.: Ueber die Bildung der inneren Nasengänge, oder primitiven Choanen, Verhandl. d. anat. Gesellsch. **145**:151, 1891.

7. Schaeffer, J. P.: Various Types of Congenital Atresias of the Nose and Their Genetic Interpretation, Tr. Am. Laryng. A. **56**:126, 1934.

8. Morgenstern, D. J.: Congenital Atresia of Postnasal Orifices, Arch. Otolaryng. **31**:653 (April) 1940.

9. Donnelly, J. C.: New Method of Operation for Congenital Atresia of Posterior Nares, Arch. Otolaryng. **28**:112 (July) 1938.

10. Anderson, C. M.: Congenital Occlusion of Choana, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1937, p. 83; J. A. M. A. **109**:1788 (Nov. 27) 1937.

11. Fraser, J. S.: Congenital Atresia of the Choana, Brit. M. J. **2**:1698, 1910.

12. Lang, J.: Ueber Choanenatresi, Monatschr. f. Ohrenh. **46**:970, 1912.

13. Ronaldson, T. R.: Note on a Case of Congenital Closure of the Posterior Nares, Edinburgh M. J. **26**:1035, 1880-1881.

infant breathed when it was held upright and the mouth was kept open. Operation, however, was delayed, and the child died. A complete autopsy was not performed. The occluding membranes were so firm that a probe was bent against them, but the instrument was finally pushed through. The exterior of the nose, the anterior nares and the hard and soft palates were normal.

In older children and adults the only symptoms are inability to breathe through the affected nostril and the presence of a persistent nasal discharge. This discharge drips from the affected naris and is characteristic, consisting of tenacious glairy mucus. It may, however, be purulent if there is infection of the nasal mucosa or of the accessory nasal sinuses.

In the majority of cases no impairment of the hearing has been reported. In both of our cases the hearing was normal. Morgenstern [8] noted that the hearing may be affected in bilateral atresia.

Several authors have stated that the voice may be considerably altered by the obstruction. In the 2 cases to be reported here there was no discernible peculiarity of the voice. The voice of the patient who was operated on was not changed after the obstruction was removed.

Anosmia has also been mentioned. In the cases in which this symptom was present, it persisted even after the patency of the nares had been restored. Anosmia was not present in either of our cases.

The treatment depends on the age of the patient, the type of obstruction and the presence of other pathologic conditions in the nares.

Respiratory difficulty in a newborn infant demands immediate investigation of the patency of the nares. Should atresia be found and the degree of respiratory difficulty be so great as to endanger the life of the patient, immediate operative intervention becomes necessary. A membranous atresia can be easily perforated and the opening enlarged, as in a case reported by Lemere,[14] that of a child 2 months of age, in which perforation with a probe, followed by dilation, sufficed for complete cure. The occluding wall, however, may be bony, as reported by Pastore and Williams.[15] In such a case the procedure is difficult and the opening necessarily small.

In the case of an older child or an adult, the character of the obstruction must, of necessity, determine the type of operative procedure. When the partition is membranous or composed of thin bone, a biting forceps and a curet may suffice for its removal. Morgenstern [8] reported the successful use of electrocoagulation in an instance in which the obstruc-

14. Lemere, H. B.: Persistent Bucconasal Membrane in the Newborn, J. A. M. A. **109**:347 (July 31) 1937.

15. Pastore, P. N., and Williams, H. L.: Congenital Occlusion of Nasal Choanae: Brief Review of Twelve Cases, Proc. Staff Meet., Mayo Clin. **14**:625 (Oct. 4) 1939.

tion was membranous. However, since it is admitted that the partition is bony in 90 per cent of the cases of congenital atresia, the use of electrocoagulation seems to be very limited. When the partition is thick and formed by dense bone, the instruments of choice are the electrically driven drill trephine and the "reaming" or "grinding" burrs. White [16] has advocated, in addition, the removal of the posterior portion of the vomer. Protection of the nasopharynx during any operative procedure is essential.

If the obstructing membrane is clearly in view during the entire operative procedure, it can be completely removed. In infants and very young children the operation will therefore as a rule be incomplete, and reoperation at a later date may become necessary.

If free access to the wall is prevented, either by a deflected septum or by any other obstacle, it may be necessary to remove this obstruction at a preliminary operation and to deal with the atresia at a later date. Pastore and Williams,[15] however, reported having performed a combined operation successfully.

Several methods of preventing the formation of occluding cicatrices have been employed. Rubber tubes (Pastore and Williams [15]) and various other obturators have been used. Donnelly [9] and Blair [17] have ingeniously planted skin grafts on the raw surfaces.

Either general or local anesthesia may be used. For young children general anesthesia has been preferred. For adults local anesthesia has proved satisfactory.

REPORT OF CASES

CASE 1.—M. P., aged 18, was first examined on Dec. 10, 1939. As long as she could remember she had been unable either to breathe through the right nostril or to blow any secretion from it. There had always been a discharge dripping from the right naris, which she described as thick and tenacious. The tonsils and adenoids were removed in childhood. When she was 8 years of age, a rhinologist was consulted. He removed a polyp from the right naris. No relief followed this procedure. There was a history of repeated attacks of bilateral acute purulent otitis media but none during the past five years.

Examination revealed a large amount of tenacious mucoid discharge in the right naris. The chamber was roomy, and after the tissues had been shrunk, an obstruction was detected immediately behind the turbinates. The middle and inferior turbinates were of normal size. Postnasal (mirror) examination showed clearly complete atresia of the choana. The obstruction was white and glistening. Probing gave the impression that it was composed of bone. The eustachian orifices appeared normal.

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16. White, L. E.: An Operation for Bony Occlusion of the Posterior Nares, Boston M. & S. J. **180**:153 (Feb. 6) 1919.

17. Blair, V. P.: Congenital Atresia, or Obstruction of the Nasal Air Passages, Ann. Otol., Rhin. & Laryng. **40**:1021 (Dec.) 1931.

Both tympanic membranes were scarred, large healed perforations being present. The hearing was within the normal range. The frontal and maxillary sinuses were clear to transillumination. No pus was seen in the nares.

On December 26 the patient was admitted to the Beth Israel Hospital for operation. Equal parts of a 5 per cent solution of cocaine hydrochloride and a 1:1,000 solution of epinephrine hydrochloride were used to obtain anesthesia. Tampons saturated with this mixture were placed in the anterior nares and in the nasopharynx.

An electrically driven drill, long and sharp-pointed, was used to make the initial opening. This was then widened by a drill of larger caliber. The bone was found to be so dense that it was perforated with difficulty and could not be removed with any of the usual biting forceps. This density of bone had not been expected. Hence, the opening when completed was only about 15 mm. in diameter. The patient could, however, breathe easily through the right nostril.

In less than one month the opening had closed completely.

On March 22, 1940 reoperation was performed. The same method of inducing anesthesia was used as in the previous operation, and a cotton tampon was left in the nasopharynx throughout the entire procedure. The site of the original opening was located, and a probe was easily pushed through. A drill trephine was used to secure a larger opening. The occluding structure was found to be composed of dense bone throughout its entire extent. By means of a large burr it was completely removed—horizontally from the septum to the lateral nasal wall and vertically from the floor of the nose to a point well above the middle turbinate. It varied in thickness from 6 mm. in its upper portion to 10 mm. in its lower portion, where it merged with the palate. There was very little bleeding throughout the operation, and no packing was necessary.

The postoperative course was uneventful. For several weeks there was a thick purulent discharge from the naris, but this cleared completely. When last seen, on Jan. 24, 1941, ten months after operation, the naris appeared normal. At the roof of the nose a small remnant of the occluding structure was seen. There was no remnant on either the septal or the lateral wall of the nose.

Case 2.—I. K., aged 55, was admitted to Montefiore Hospital on Aug. 6, 1939 with the diagnosis of carcinoma of the prostate with metastases.

On March 28, 1940, consultation was requested because it was noticed that the patient could not breathe through the right naris. He stated that he never had been able to breathe through the nostril. He had sustained a nasal fracture when he was a young man, but he insisted that he could not breathe at all through the right nostril prior to this. This inability to breathe through one nostril did not cause him any inconvenience, and he had never consulted a physician about it.

Examination revealed a marked deflection to the right of the anterior portion of the septum. Crusts and pus were present in the right naris. Postnasal examination showed complete atresia of the right posterior choana. There were two minute indentations near the center of the occluding membrane, which were thought to be perforations through it. The eustachian orifices appeared normal. The hearing in both ears was within the normal range.

Roentgen examination by Dr. A. J. Bendick showed the frontal sinuses to be of normal size and illumination. Both antrums were slightly clouded, the right showing the presence of cysts. The right sphenoid sinus was smaller than the left. The entire right naris appeared "cloudy," but a diagnosis of atresia could not be

made from the roentgenograms. A contrast medium, as suggested and demonstrated by Donnelly,[9] was not utilized.

On October 29 the patient died of the prostatic malignant condition.

Postmortem Examination of the Nose and Accessory Nasal Sinuses.—Macroscopic: Except for a shallow but not unusually small right sphenoid sinus, all the sinuses were of normal size. The frontal sinuses were normal. The mucosa of the right antrum was edematous in appearance, but the sinus contained no free

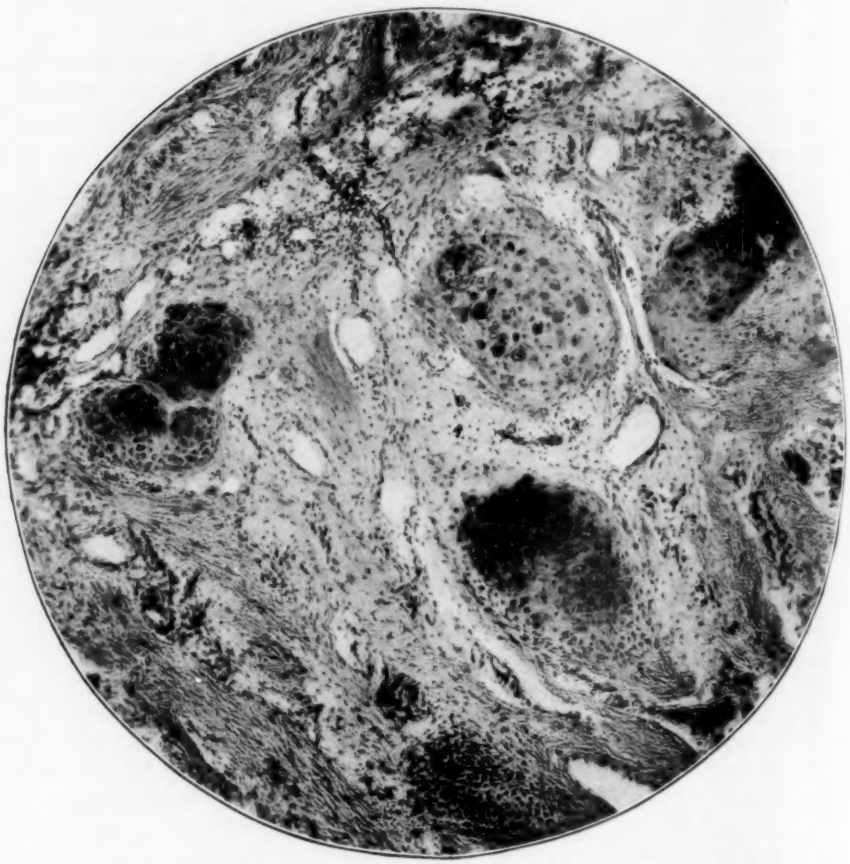

Fig. 1 (case 2).—Islands of cartilage near the middle of the occluding wall (hematoxylin and eosin stain; × 120).

fluid. There were several large mucous membrane cysts in the antrum, filling about one third of the cavity. The mucosa of the left antrum was thickened and edematous in appearance, but no free fluid was present. The mucosa of the right sphenoid sinus was normal in appearance. The mucosa of the left sphenoid sinus was edematous. The ethmoid cells of both sides were normal.

The occluding wall was exposed by removing the anterior portion of the hard palate. The posterior choana of the left side was normal. The right naris ended

in a smooth glistening grayish cul-de-sac, concave on the nasal side. Toward the septum and the floor of the nose the occluding membrane was bony, and toward the lateral wall and the roof of the nose it was membranous. That portion of the septum between the anterior deflection and the atretic obstacle was in the midline. The occluding wall was 10 mm. in thickness at its lower part, where it was composed of bone, and 8 mm. in thickness above, where it was membranous. The mucosa of the anterior surface of the wall was continuous with that of the septum and of the lateral nasal wall.

The turbinates of the left naris were normal in size and appearance. On the right side the anterior two thirds of the turbinates were of normal size. The posterior thirds of both middle and inferior turbinates were much smaller than normal,

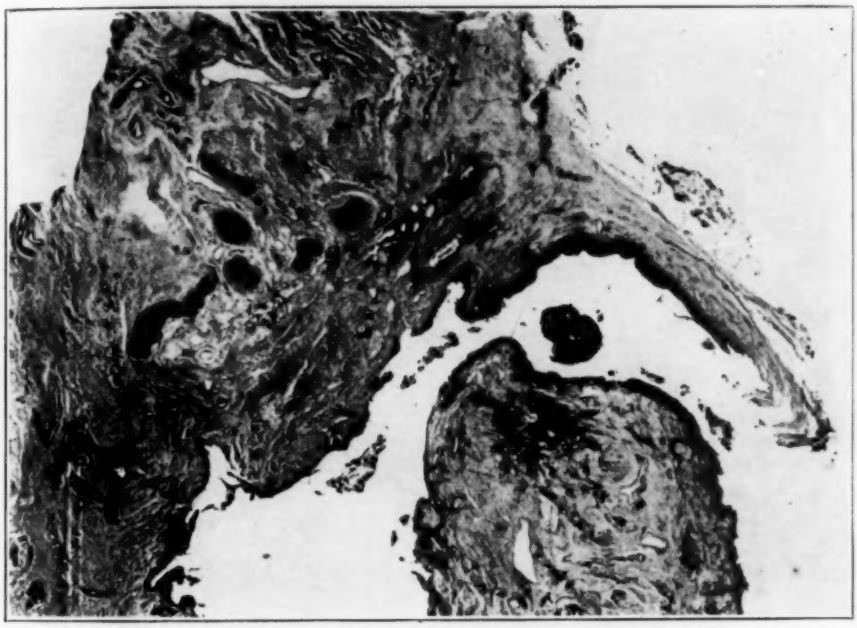

Fig. 2 (case 2).—Foramen through the occluding wall (hematoxylin and eosin stain; × 60).

and instead of having well formed tips, the posterior ends of both turbinates blended into the lateral nasal wall, leaving only a small bulge. That portion of the right naris behind the anterior deflection of the septum appeared roomier than the corresponding portion of the left naris. The occluding wall was situated almost immediately behind the undeveloped tips. The right eustachian orifice was a few millimeters behind the wall and was normal in appearance.

Microscopic: The entire mass containing the occluding wall was removed and decalcified. On account of the decalcification the mucosa was severely denuded in places, but a sufficient amount of it remained for positive identification.

The mucosa of both antrums and the left sphenoid showed marked edema. Mucous membrane cysts were present in the right antrum.

Numerous sections were made at different levels of the atretic partition. The nasal surface throughout was covered with pseudostratified columnar epithelium, which blended with the mucosa of the septum and the lateral nasal wall. The posterior surface was covered with stratified squamous epithelium. Unfortunately, the mucosa of the septum posterior to the atresia was desquamated.

The uppermost part of the atretic partition, between the two layers of mucous membrane, was composed of firm connective tissue and contained occasional glands.

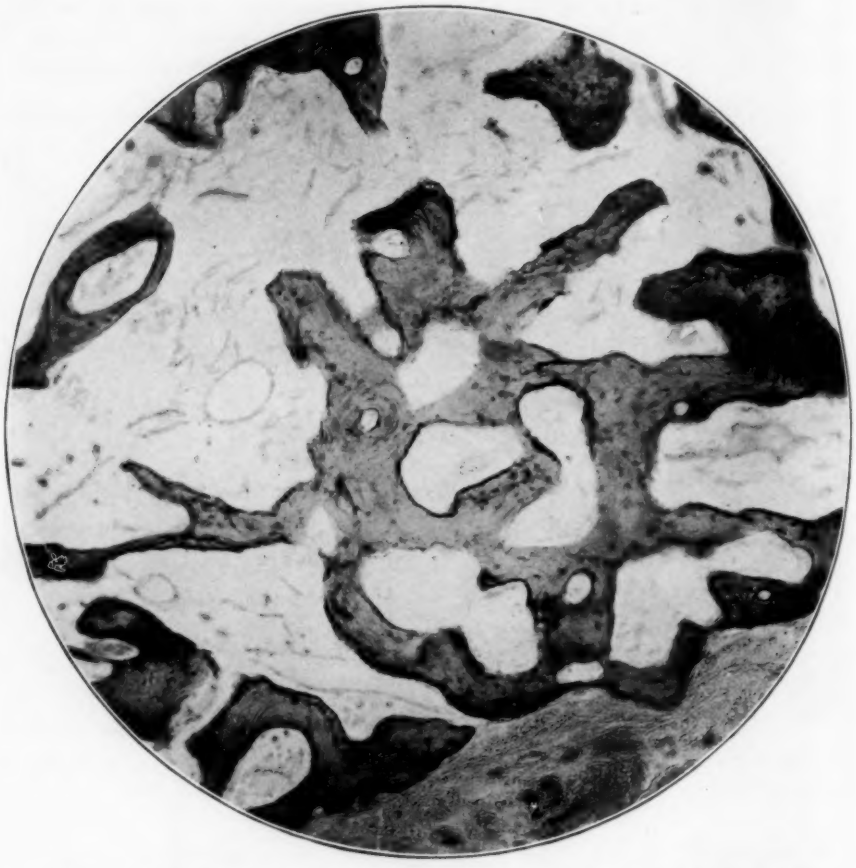

Fig. 3 (case 2).—Bone from the lower portion of the occluding wall (hematoxylin and eosin stain; × 120).

At a lower level the membrane was composed mainly of connective tissue, in which were several islands of hyaline cartilage, some of which were partially calcified. There were also a few glands and some medium-sized blood vessels. In some areas there was a moderate amount of round cell infiltration.

Near the nasal wall, in the region of the inferior turbinate, were numerous venous channels of the type found in the posterior portion of the inferior turbinate.

Through the center of the atresia, at the level of the attachment of the inferior turbinate, a tortuous foramen was found, which extended completely through the

membrane. The lining epithelium toward the naris was pseudostratified columnar. Toward the center of the canal it changed to a "transitional" type and in the posterior portion to stratified squamous epithelium.

The lower part of the occluding wall was composed of bone. Between the bone and the surface epithelium was a layer of dense connective tissue containing a few venous channels lined with a single layer of endothelial cells. Very few glands were present. The bone was of the cancellous type, identical with the spongy bone normally found in the vomer.

The posterior ends of the turbinates of the right naris were markedly fibrous and contained numerous venous channels. The middle and inferior turbinated bones were, in this part, thinner and smaller than normal. The soft palate was normal.

Of interest in our pathologic observations was the presence of stratified squamous epithelium on the posterior surface of the occluding wall and extending part of the way into the foramen through it.

The normal lining membrane of the entire nares and the anterior part of the upper surface of the soft palate are lined with pseudostratified ciliated columnar epithelium. The only parts of this region which are lined with squamous epithelium are the posterior portion of the upper surface of the soft palate and the posterior wall of the nasopharynx, except in the region of the vault.

Maximow and Bloom [18] stated that with age the ciliated epithelium may be replaced by stratified squamous epithelium over large areas. However, it is possible that this squamous epithelium was a part of the "under" surface of the bucconasal membrane of Hochstetter and hence was derived from the oral mucosa.

In addition to those in the cases of Otto and Roederer, postmortem examinations in cases of congenital atresia have been reported by Luschka,[19] Bitot,[20] Kundrat,[21] Berblinger,[22] Hamilton,[23] Heymann [24] and Fraser.[11] In the case reported by Hamilton the specimen was macerated to show the extent of the condition. Fraser quoted Hamilton's description as follows:

The septum was straight; bony choanal plates are diaphanous in the center; the bony plates are attached in front and below at the junction of the superior

18. Maximow, A. A., and Bloom, W. A.: A Textbook of Histology, ed. 2, Philadelphia, W. B. Saunders Company, 1938.

19. Luschka, H.: Ueber angeborene Atresi der Choanen, Virchows Arch. f. path. Anat. **18**:168, 1860.

20. Bitot: Atrésie ou obturation congénitale des orifices postérieures des fosses nasales, Arch. de tocol. **3**:554 (Sept.) 1876.

21. Kundrat, H.: Die Arhinencephalie als typische Art von Missbildung, Graz, Leuschner & Lebensky, 1882.

22. Berblinger, W.: Der angeborene Verschluss der Choanen, Arch. f. Laryng. u. Rhin. **31**:632, 1917-1918.

23. Hamilton, T. K.: Bony Occlusion of the Choanae, J. Laryng. **20**:172 (April) 1905.

24. Heymann: Präparat von knöcheren Choanalverschluss, Berl. klin. Wchnschr. **15**:4, 1904.

maxillary and palate bones; at both sides to the internal pterygoid plates; nasally to the posterior margin of the vomer; behind and above to the basilar process of the occipital bone. The septum at its base projects behind the plane of the bony plates.

Fraser's own case, one of atresia on the right side, was fully described. He gave measurements of the nares, comparing the two sides, showing that the hard palate on the affected side was higher than the normal and that the nasal cavity on that side was much narrower. The posterior end of the right inferior turbinate was absent or at most was represented only by a slight projection on the outer wall of the nose. The right middle turbinate was poorly developed as compared with the left. Fraser also described a pit or canal in the middle of the bony plate, which ran forward, apparently into the nasal septum. The right sphenoid sinus was considerably smaller than the left.

In regard to microscopic examination, Luschka merely mentioned that the nares were lined with ciliated epithelium. Berblinger, who made serial sections of the occluding wall in a case of bilateral atresia, noted that this was composed of bone containing marrow spaces. In our case 2 the bone was of the cancellous type. Both Berblinger and Fraser noted desquamation of the epithelium, and Fraser, in addition, mentioned considerable round cell infiltration in the olfactory region. Berblinger attributed this desquamation to a "desquamative catarrh," which he claimed is always present in cases of atresia. Our experience has been, however, that epithelial desquamation is incidental to the process of decalcification.

COMMENT

Two cases of congenital atresia of the posterior choanae are reported. Both patients were adults. One was operated on, and the other came to post mortem examination.

The findings in practically all of the reported cases agree in all major points. Variations occur only in minor details.

The condition should be suspected in cases of asphyxia neonatorum and in infants with complete nasal obstruction.

We believe that if it is possible to remove the entire occluding membrane, obstructive cicatrices will not form.

7 West Seventy-First Street.

897 Park Avenue.

FURTHER OBJECTIONS TO THE ACCEPTED INTERPRETATIONS OF COCHLEAR MECHANICS

A. G. POHLMAN, M.D.
LOS ANGELES

The late Gustave Alexander,[1] in a paper published in 1930, offered a bit of valuable advice to those investigating problems in audition. This advice is well worth repeating, even if Alexander himself did not follow it. He said:

> We must avoid overpowering the facts. Our theory must place us in a position to readily explain all the facts derived through anatomical study and through clinical observations. The application of facts, correct in themselves, toward passing judgment on theories of hearing, have been subject to repeated error.

It is universally accepted that the auditory cells react to a physical stimulus resulting from sound vibrations and that this response to pressure is essential to the activation of the applied nerve terminals. There are at least two ways in which this may come about. The accepted interpretation assumes that the auditory cells are nonspecific in their frequency reactions because all of these cells are structurally similar. It is further assumed that the specific stimulus to these cells is provided by transverse vibrations of the basilar membrane, which acts as a mechanical harmonic analyzer. The harmonic analysis is dependent, in turn, on a selective vibrational pattern which separates the complex sound into its several components. The vibrational pattern is impressed on the applied auditory cells, and the brain translates this pattern into terms of the pitches heard. This, briefly, is the "place" theory of hearing. A second explanation, proposed years ago by me,[2] is based on the assumption that the auditory cells are specific in their reactions to the pulsating pressures applied to them directly through the labyrinth liquid. While this interpretation does not imply denial of the possibility of a "place" theory, it does imply that the assumed transverse vibrations in the basilar membrane are not essential to audition. There are serious physical and

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From the Department of Otolaryngology, University of Southern California School of Medicine.

Assistance was given to the research on hearing through a grant-in-aid by the Carnegie Corporation.

1. Alexander, G.: Monatschr. f. Ohrenh. **65**:1 and 173, 1930.
2. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **40**:717, 1931.

physiologic objections to the accepted interpretation, but thus far none has been proposed to the second explanation.

The fact that the auditory cells are structurally similar throughout the length of the end organ is not necessarily a criterion by which all may be adjudged similar in function and therefore not specific in their reaction to a frequency analysis. Inasmuch as it is not known what a cell which reacts to sound vibrations would look like to start with, it might be difficult to correlate structural variations with functional differences, even if such variations could be seen. One may, however, reason by analogy. The fact that the retinal neuroepithelium shows no structural differences does not make it necessary to assume that it is not specific in its reactions to color. Neither can color blindness be successfully explained on the basis of a microscopic investigation of dead material. Similarly, the olfactory epithelium shows no evidence of structural differences throughout its extent. This fact does not make it necessary to assume that its activation is an indirect one and that some structure extrinsic to these cells is responsible for the analytic capacity displayed in the discrimination between odors.

The accepted interpretation is based on the assumption that transverse vibrations in the cochlear duct, specifically in the basilar membrane, are essential to audition. It was first proposed by Weber,[3] in 1851. Weber found that when the stapes of a specimen was displaced into the oval window, the round window membrane bulged outward into the tympanic cavity. He argued that a similar reaction to sound vibrations would also come about. He postulated that the labyrinth liquid must be regarded as an incompressible medium and that the dense otic capsule presents an inelastic container. Under these physical conditions the only possible displacement in the labyrinth liquid would be a mass shifting of the liquid dependent on interaction between the two windows. This conception of the mechanics could be directly correlated with what was then known of the cochlea from the standpoint of morphology. Thus, when the pressure exerted by the air sound pushed the stapes into the oval window, the liquid of the scala vestibuli would be displaced toward the helicotrema, while that of the scala tympani would move toward the compensation area essential to this shifting, the round window membrane. The displacement of the liquid on either side of the membranous cochlear duct would accordingly result in transverse vibrations in the basilar membrane and in activation of the nerve terminals distributed throughout the length of this structure.

Thus, the doctrine of transverse vibrations in the basilar membrane came into being and was applied to the indirect activation of the end

3. Weber, E. H.: Verhandl. d. k. Gesellsch. d. Wissensch. zu Leipzig, 1851, p. 29.

organ when it was later discovered that the nerve terminals were distributed to the auditory cells and not to the basilar membrane. The accepted interpretation is clearly visualized in a diagram by von Bekesy,[4] reproduced here as figure 1 *A*. That this interpretation is widely accepted is indicated in a recent statement by Davis,[5] who as co-author of the textbook on hearing by Stevens and Davis,[6] spoke as one well oriented in the subject:

> I feel that with the possible reservations as to very low tones—so low that the sense of hearing merges over into the sense of vibration—we can confidently say that the sensation of pitch is determined by the position of the vibratory pattern on the basilar membrane and not by the frequency of the nerve impulses in the auditory nerve.

It is indeed a strange commentary on modern methods of research that the seemingly obvious explanation proposed by Weber in 1851 should have been accepted without previous confirmation of the physics on which it is based or even the establishment of the described sequence

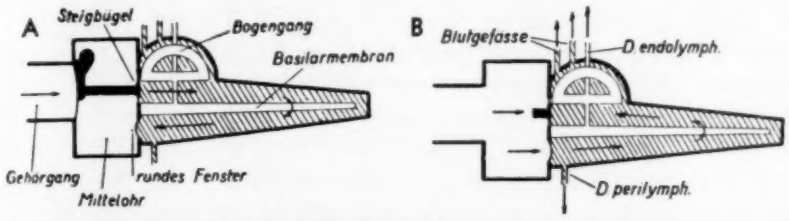

Fig. 1.—*A* shows the accepted reaction to sound in the internal ear when the sound conduction apparatus is functional. Here the round window membrane compensates for displacement in the stapes. *B* shows how pressure on the round window in the absence of a drum membrane and outer ossicles is compensated in the venting system described by von Bekesy, with reversal of normal phase in the vibrations at the cochlear duct.

of reactions as fact. Waetzmann,[7] an eminent physicist who has been interested in problems of audition for many years, categorically denied that the cochlear liquid may be regarded as an incompressible medium. He said:

> One must, however, understand clearly that in so far as a detailed consideration is concerned, a liquid cannot be regarded as a completely incompressible

4. von Bekesy, G.: Akust. Ztschr. **1**:13, 1936.

5. Davis, H.: Laryngoscope **50**:747, 1940.

6. Stevens, S. S., and Davis, H.: Hearing: Its Psychology and Physiology, New York, John Wiley & Sons, Inc., 1938.

7. Waetzmann, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellenger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1926, vol. 11, p. 678.

medium, because otherwise variations in density would be impossible and the rapidity with which a pressure applied to one area would be conducted would be infinitely great.

When this denial of incompressibility of the cochlear liquid is coupled with the use of the elasticity in the otic capsule to explain the hearing of bone-conducted vibrations, it is obvious that both premises postulated by Weber present physical impossibilities. Further, the contention that interaction between the two windows is essential to audition has never been convincingly demonstrated. Still further, under conditions of stapes fixation the assumed mass shifting in cochlear liquid must be suppressed and all transverse vibrations in the basilar membrane eliminated. All persons with stapes fixation should, therefore, be quite deaf unless there is some way to produce the transverse vibrations other than the one proposed by Weber.

Von Bekesy,[4] a physicist and in my opinion one of the most ingenious of all investigators in this field, has proposed an explanation which may be directly applied to the problem of producing transverse vibrations in the basilar membrane under conditions of stapes fixation. Von Bekesy examined subjects in whom the drum membrane and outer ossicles are wanting, and his interpretation of the mechanics of the cochlear reactions under these conditions is clearly indicated in figure 1 *B*. The diagram shows that when the sound pressure is applied to the medial tympanic wall the stapes is fixed but the round window membrane is displaced into the scala tympani. According to the accepted interpretation, which is based on interaction between the two windows, this would be impossible. Von Bekesy, therefore, looked for other ways in which the cochlear liquid might be shifted and indicated these areas of compensation as the ductus endolymphaticus, the ductus perilymphaticus and the blood vessels. Obviously, the ductus endolymphaticus presents a vent on the upper side of the basilar membrane, while the ductus perilymphaticus connects the scala tympani with the subarachnoid space projected out over the glossopharyngeal nerve. It is also true that the vascular system related to the scala vestibuli, the vestibule and the semicircular canals is more extensive than that of the scala tympani. Von Bekesy interpreted this to mean that the upper venting system is more effective in permitting displacements of liquid from the spaces related to the upper surface of the basilar membrane than is the lower venting system, connected with the scala tympani. Accordingly, the inward displacement of the round window not only becomes possible but produces the assumed vibrations in the basilar membrane in a phase opposite to normal. This is clearly indicated by comparing the direction of the arrows in figures 1 *A* and 1 *B*. Von Bekesy did not suggest what forces the displaced liquid back again when the pressure phase of the sound pulse is followed by the

rarefaction phase, but, for that matter, none of the exponents of the doctrine of transverse vibrations in the basilar membrane even mention the restoring forces essential to this reaction.[8] Von Bekesy arrived at this interpretation to explain the confusion of the patient who is attempting to locate the direction of a sound source. Next, he applied a cotton pellet prosthesis to the stapes area and found that the spatial recognition was altered. He argued that the prosthesis produced effective vibrations of the stapes and that the phase of reaction in the basilar membrane was reversed to the normal. Davis also referred to the possibility of a similar phase reversal in the vibrations of the basilar membrane:

> Ordinarily, if the eardrum is intact, the tympanic membrane is periodically attracted by electro-static force to the promontory opposite to it. Unless a direct-current polarizing voltage is also applied, the subject hears a tone an octave above the frequency of the alternating current, since the eardrum is attracted to the promontory twice in each cycle, at both the positive and the negative peak. If, however, the eardrum is missing and the voltage is applied to the round window, the subject usually hears a tone corresponding to the actual frequency of the current. We are not quite so sure as to the mechanism in this situation. The most plausible guess is that whatever mechanism in the ear normally generates electricity and produces the aural microphonics when sound agitates the basilar membrane is being operated "in reverse," so to speak, and causes the basilar membrane to vibrate in response to the alternating current applied to the ear. We can sum up this explanation by saying that this particular *electrophonic* effect is the inverse of the familiar *aural microphonic* effect.

At a meeting of the American Physiological Society in 1935, I [9] presented objections to the Weber interpretation, and at a meeting of the Collegium oto-rhino-laryngologicum in 1937 I [10] amplified these objections to include von Bekesy's explanation.

It was pointed out that a cotton pellet prosthesis applied to the stapes is effective only when the ossicle is sufficiently movable to accept the vibrations. Inasmuch as the stapes reacted to the cotton pellet in von Bekesy's case, there is no reason to suppose that it did not vibrate to the air sounds applied to it. The effectiveness of the pellet prosthesis to low tones is rarely more than 10 decibels. Accordingly, a less effective pellet might produce the result if the assumed upper vents would compensate for the displacement at the oval window while the lower vent accommodated all displacement at the round window. Under these conditions complete cancellation of all transverse vibrations in the basilar membrane would come about, and the patient should be quite deaf. However, no cases have been reported in which the application of a cotton pellet to the stapes has resulted in deafness, and in my experience the pellet prosthesis either enhances the air acuity or is without effect.

8. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **48**:148, 1939.
9. Pohlman, A. G.: Am. J. Physiol. **113**:106, 1935.
10. Pohlman, A. G.: Acta oto-laryng. **26**:162, 1938.

It was also brought out that stapes fixation is extremely common in patients with progressive deafness. What must be the experience of these patients according to the von Bekesy interpretation? If transverse vibrations in the basilar membrane are essential to audition, as soon as the fixation has progressed to the point that the assumed upper vents would compensate for stapes displacements, the patients must be deaf. This time would coincide exactly with the time of the assumed phase reversal. Later, as the stapes became more fixed, a certain return in the hearing acuity would be expected. This return, however, has never been reported. If it occurs, the fact should become common knowledge. Therefore, one who agrees with the accepted interpretation must assume that all patients with stapes fixation are deaf, and one who agrees with the von Bekesy proposal must assume that they become deaf before the stapes is fixed. Again I repeat the advice of Alexander: "We must avoid overpowering the facts."

Polvogt and Crowe [11] reported 6 cases of scala communis cochleae:

> There was a dehiscence in the bony septum between the middle and apical turns in six of the seventeen cochleae. This anomaly was described by Alexander who called it "Scala communis cochleae." According to some theories of hearing, a scala communis which establishes a free communication between the scala vestibuli and the scala tympani should impair the hearing. The location of the defect in our cases should theoretically impair the hearing for low tones but the audiometric test showed normal threshold acuity for all tones in every patient.

Since 1937 I have had the opportunity of examining a number of patients with auditory disability and have paid particular attention to those in whom the drum membrane and outer ossicles were wanting. This was done with the idea of replacing the lost middle ear apparatus with a diaphragm-rod prosthesis patterned after the sound conduction apparatus in birds.[12] It is essential to the proper operation of this type of prosthesis that a yielding contact area for the rod be definitely located in the medial tympanic wall. The information was gained through the use of a simple acoustic probe. This probe consists of a stalked diaphragm of mica supporting a long slender stem of bamboo or wire, suitably tipped. The diaphragm is held by the stalk, and the stem is introduced through a speculum. The examiner explores the entire medial tympanic wall by touching it lightly here and there while he keeps talking. When a yielding area related to the perilymphatic space is contacted, the patient will immediately declare that the examiner's voice has suddenly become loud and deformed. The loudness is due to the vibrations in the mica diaphragm conducted through the rod to the

11. Polvogt, L. M., and Crowe, S. J.: Ann. Otol., Rhin. & Laryng. **46**:579. 1937.

12. Pohlman, A. G.: J. Morphol. **35**:229, 1921.

yielding area. The deformation in the voice is dependent on the low free period of reaction in the probe. The acoustic probe always produces this positive response when applied to the movable oval window area or to the round window, where this is accessible. The response is always negative when the probe contacts a fixed stapes or the promontory.

It was observed that the acuity of hearing for air-borne sounds was similar in patients with complete conduction deafness whose middle ear apparatus was intact, provided the drum membrane was not diseased, and in patients in whom the drum membrane and outer ossicles had been lost, whether the oval window was fixed or was movable as determined by the acoustic probe. Necessarily, this was observed only in cases in which the perceptive apparatus was relatively normal, as determined by the bone acuity curve. The air acuity in 2 characteristic cases is presented in figure 2 *A*. In case 1 repeated exploration of the medial tympanic

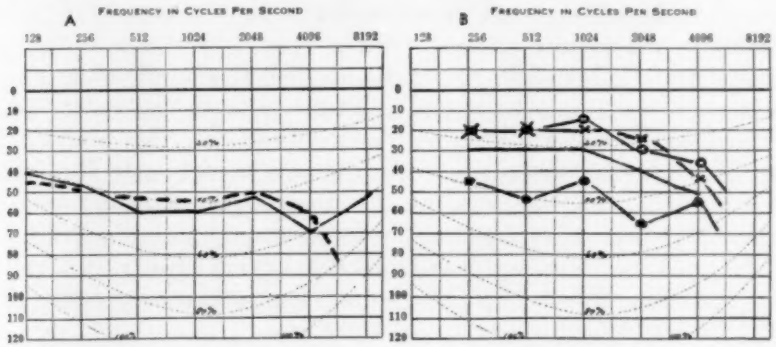

Fig. 2.—*A*, audiogram showing hearing by air conduction in 2 cases. The solid line represents the hearing level in case 1; the dash line, that in case 2. *B* (case 2), curves resulting from experimental contact of a telephone probe with various structures. The line broken by circles represents the curve from contact with the oval window; that broken by crosses, the curve from contact with the round window; the solid line, the curve from contact with the promontory, and the line broken by dots, that from contact with a tooth.

wall failed to reveal any yielding area as evidenced by a positive reaction to the acoustic probe. In case 2, on the contrary, the probe demonstrated a round window area accessible to contact and an oval window both of which gave equally positive reactions. It will be noted that the air acuity curves in figure 2 *A* are quantitatively similar except at the frequency 8192, where in case 2 perception deafness was superimposed on the condition.

The patient in case 2 was extremely cooperative and submitted to further experiment. A telephone probe was constructed by affixing a slender rod of bamboo to the vibrating plate of a miniature bone receiver. The rod was tipped with a small ball of dental stent. The patient

introduced the probe himself, and a tone was exhibited to permit him to make effective contacts at the oval and round windows. The contact on the one or the other window was determined by the direction taken by the rod. Acuity curves were taken from the oval and round windows and were compared with those obtained through contacts with the promontory and the upper lateral incisor tooth on the same side.

The result is indicated in figure 2 *B*. The curves are similar in form within limits of a probable experimental error. The acuity curves developed by contact of the probe with the oval and the round window were practically identical. When contact was made on the promontory, the hearing was consistently less sensitive, and the intensity required when contact was made with the tooth was considerably greater. Obviously, the threshold for hearing by direct bone conduction through the promontory was only somewhat higher than that for contact with the oval and the round window. This is consistent with a previous report.[13] The discrepancy is probably variable, depending on the condition of the lining membrane and on the thickness of the bone. Inasmuch as the two window areas appeared to be equally yielding to the vibrations of the telephone probe, it may be assumed that they also yielded about equally to the direct pressures applied by the air vibrations. This would mean that all interaction between the two windows, according to the Weber interpretation, would be suppressed with resultant elimination of all transverse vibrations in the basilar membrane. If transverse vibrations in the basilar membrane are essential to audition, this patient should have been deaf to both air-conducted and bone-conducted sounds.

Davis stated:

> The sound wave is a wave of compression traveling through the bony structure of the skull, and as it passes through temporal bone it squeezes fluid from the semi-circular canals, vestibule and scala vestibuli toward the round window, which is the most flexible and yielding portion of the labyrinth wall, particularly if the stapes is ankylosed. But if the ossicular chain is freely movable, the inertia of the ossicles themselves is sufficient to set up movement in the perilymph as the skull moves to and fro or rotates from the impact of the bone conduction receiver.

Accordingly, in case 1, in which the oval window was fixed, the bone acuity should have been greater than normal. It was not. And in case 2, with the two windows equally movable, the motional reactions in the basilar membrane should have been materially reduced and the bone acuity should have been less than normal—conditions which were not observed. According to von Bekesy's explanation, the air acuity in case 1 should have been greater than that in case 2, whereas, if there was a difference, the reverse was true. I again repeat the advice of Alexander not to "overpower the facts."

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13. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **39**:927, 1930.

Recently, Lempert [14] published an extensive report on the results of his operation. He selected patients in whom the stapes was fixed and who, according to the accepted interpretation, should be quite deaf. When the perilymphatic space of the external semicircular canal was exposed, the patients immediately declared an increase in air acuity. This was confirmed by audiometric evidence, and Lempert emphatically stated that the bone acuity was not affected. According to the accepted interpretation, the opening of the external semicircular canal should have no effect in cases of stapes fixation. This would be true, for in stapes fixation the assumed mass displacements are suppressed, because only the round window is movable. When the lateral semicircular canal is opened, the pressure in the air vibration is applied to both fenestra and round window at the same time and all possibility of interaction should be canceled out. According to the von Bekesy explanation, opening the external semicircular canal should materially reduce the acuity of hearing. This would be true, because the liquid displaced by the pressure on the fenestra would be compensated in the upper vents, while the pressure at the round window would be taken up by the lower vent. According to both the accepted interpretation and that of von Bekesy, the bone acuity should be materially reduced, because both the round window and the fenestra permit liquid displacement. The fact that Lempert's cases do not demonstrate a lack of enhancement to air acuity and a reduction in bone sensitivity is unfortunate for one who wishes to prove the theory that transverse vibrations in the cochlear duct are essential to hearing.

There is, however, something more to be explained. Why did the patient in case 2, with two movable windows, one on either side of the cochlear duct, not hear as acutely as did a patient who was operated on successfully by Lempert and in whom there were also two windows, the fenestra and the round window, one on either side of the cochlear duct? The only explanation which suggests itself, and this is purely conjecture, is that the fenestral opening delivered the sound pressures to the labyrinth liquid more effectively than did either the round window or the oval window in case 2. This suggestion appears to be borne out by a case reported by Lempert, in which the fenestral opening was covered with a relatively heavy connective tissue membrane, with loss of the conferred acuity. It must be remembered that the degree of a mass displacement assumed to occur through interaction between the two windows is determined not by the more yielding window but by the more fixed window. The important contribution of these fenestra-

14. Lempert, J.: Endaural Fenestration of External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **31**:711 (May) 1940.

tions is that the patients hear more acutely, even if the assumption of transverse vibrations in the basilar membrane and all theories based on this interpretation may be abandoned. The loss would not be serious, because none of the theories of audition thus far proposed fits the established facts.

I quote from a more complete analysis of this problem, published in 1933:[15]

> The "proposed mechanics" suggests that the auditory cells react directly to the vibrations in the liquid in which they are immersed, and that the function of the middle ear apparatus is to drive the air vibrations into the water and thus overcome the acoustic insulation. The morphology, which has hitherto been explained on the basis of sound conduction and sound analysis, may be more successfully correlated with the developmental pattern and with a shock absorbing function in both middle and internal ear. If this explanation is correct, then the confusion in the interpretation of the auditory apparatus is dependent on misinterpretations and misdirected correlations.

15. Pohlman, A. G.: J. Laryng. & Otol. **48**:156, 1933.

SPECIFIC VACCINE THERAPY IN TREATMENT OF SINUSITIS

MYER SOLIS-COHEN, M.D.
PHILADELPHIA

In every case of sinusitis, as of all other diseases, it is necessary for the physician to determine whether he may safely entrust recovery to Nature alone or whether he must come to her aid with the resources of his art. He therefore must understand, to the extent that present knowledge permits, the normal operations of the human body when infected with pathogenic organisms.

Whenever these organisms gain entrance into the tissues, their toxic products stimulate the tissues to manufacture antibodies for the purpose of destroying the microbes and neutralizing their toxins. Should the bacteria overcome this natural immunity, the subsequent arrest of their growth comes not from exaltation of the bactericidal powers of the local tissues but from antibacterial substances elaborated in the uninfected tissue.[1] The scientific exploitation of this protective mechanism for therapeutic uses is the essential feature of the treatment of bacterial disease by vaccines.[2] Inoculating the specific bacteria at a distance from the primary focus of the bacterial growth incites the chemical machinery of the body to immunizing response, the tissues in the region of the inoculation reacting by production of substances which combine with, render inert and break down the bacterial protoplasm. The surplus of these antibacterial substances finds its way in the blood stream to the focus of infection, where it brings aid to the defensive forces of the organism, which before were ineffectually combating the infective bacteria. The victory which the uninfected tissues have won over the microbes of the vaccine will in this way lead to a victory of the infected tissues over the microbes they have to combat.[3]

Read before the Section on Otolaryngology of the College of Physicians of Philadelphia, Nov. 20, 1940.

1. Adami, J. G.: The Basal Principles of Vaccine Therapy, J. A. M. A. **54**:1922 (June 11) 1910.

2. Wright, A. E.: The Principles of Vaccine Therapy: Bacterial Inoculation Under the Guidance of the Opsonic Index, J. A. M. A. **49**:479 (Aug. 10); 567 (Aug. 17) 1907.

3. Wright, A. E.: Vaccine Therapy: Its Administration, Value, and Limitations, London, Longmans, Green & Company, 1910.

Vaccine treatment is a broad term for the inoculation of dead bacteria or some fraction or product of them. A nonspecific vaccine has no relation to the type of micro-organism causing the infection. In specific vaccine therapy the same type of organism that is believed to be the etiologic factor is employed. An autogenous vaccine consists of microbes obtained from the site of the patient's infection.

In employing a stock vaccine some physicians pay no regard to the type of organism infecting the patient, while others endeavor to determine this by culture. Vaughan [4] remarked that the stock streptococcus or staphylococcus vaccine put out by the pharmaceutic supply houses may have been obtained five years before from a group of patients in the Middle West and that there is no reason that this vaccine should be appropriate five years later for a patient in the Southeast or even in the Middle West.

Adami [1] expressed the belief that more constant results are to be gained by the employment of dead cultures of the particular strain of pathogenic bacteria causing the infection in and isolated from the patient than by the use of laboratory stock cultures. He stated that such a vaccine, liberating a particular order of toxins and other bacterial products, would more surely stimulate the production of the particular order of antibodies necessary to counteract and destroy the infective micro-organisms.

Many different preparations are included under the term vaccine—whole organisms, the aqueous extract of the organisms, bacterial filtrates and a combination of the microbes and their soluble or exogenous toxins, known as vaccine filtrates. I prefer the last, inasmuch as they stimulate the production of both bactericidal and antitoxic substances.

Some physicians employ vaccines to prevent the occurrence of "colds," with their accompanying sinusitis. The object of the prophylactic use of vaccines is to raise resistance in an uninfected person against organisms which may subsequently attack him. Prophylactic vaccines are commonly employed in the prevention of diseases of known origin, such as typhoid fever, and are administered in very large doses. For the prophylaxis of "colds" the commercial laboratories supply various combinations of those bacteria which are thought to be capable of causing "colds," none of which contains the virus which many believe to be the etiologic factor in the "common cold."

What is frequently mistaken for the "common cold" is an acute exacerbation of chronic latent sinusitis that occurs when bodily resistance is lowered from any cause. Vaccine administered in cases of this type

4. Vaughan, W. T.: Allergy and Applied Immunology, St. Louis, C. V. Mosby Company, 1931.

is not strictly prophylactic and, if potent and given in the large prophylactic doses commonly employed, may cause severe and harmful reactions should it contain the same organism that is present in the sinus.

ACUTE SINUSITIS

In acute sinusitis the patient's tissues are endeavoring to manufacture antibodies faster than the bacteria can multiply and in greater amount than the infecting organisms can produce. Some physicians employ vaccines in treating this condition, although all that may be required is to promote the physiologic factors which increase bodily resistance, such as rest, proper food, proper elimination and avoidance of chilling, with mild local treatment to promote drainage and possibly some medication. The addition of injected antigens to those being produced by the infecting bacteria may in some cases overwhelm and paralyze the immunizing mechanism.

One ordinarily cannot wait to make an autogenous vaccine in cases of acute sinusitis but must use a stock vaccine. In making the former it usually is not difficult to obtain the etiologic organism, which, as a rule, is the one that predominates in a culture of material taken from the infected area.

I have had little personal experience with vaccines in treatment of acute sinusitis.

CHRONIC SINUSITIS

Subacute and chronic sinusitis present a different situation from that obtaining in the acute condition. In chronic infections the lowered local resistance, which favored the acute attack, persists to a greater or less degree, with the result that the infected tissues fail to return to normalcy. According to Zinsser,[5] failure of chronic lesions to heal may be due to an insufficient supply of circulating antibodies or opsonic or bactericidal substances. By artificially stimulating the production of antibodies with injections of vaccine, therefore, the physician can assist nature in combating chronic sinusitis.

If, however, a vaccine is to be potent and really specific it must contain the infecting organism. Unfortunately, most bacteriologists, without any scientific justification and merely by drawing an analogy from the bacterial situation in acute infection, regard as the etiologic factor in a subacute or chronic infection those bacteria which are present, or which predominate, in an ordinary or in a dextrose–brain broth culture of material from the infected area. Some, with even less scientific accuracy, decide from their general knowledge, or simply

5. Zinsser, H.: Infection and Immunity, ed. 3, New York, The Macmillan Company, 1923.

guess, which of the organisms present is the infecting agent. Yet it has been shown that the causative germ may not be present in the culture [6] or may be overgrown by contaminating organisms and that the predominating microbe may not even be pathogenic for the patient.[6e]

A few bacteriologists and immunologists realize that it is important to know which of the organisms present is infecting, or is capable of infecting, the patient. For determining the pathogenicity of bacteria for their host a number of tests have been employed, most of which are unreliable.[7] The best criterion for pathogenicity is the ability of an organism to grow in the blood of its host, based on the fact that an animal often is able to remove from its blood stream germs that have gained access to it.[8] Various methods of testing hemobactericidal power have been devised, which employ the patient's blood serum, his defibrinated, uncoagulable or coagulable whole blood or an agitated mixture of his leukocytes and serum.[7] The best and simplest of these is the pathogen-selective, or Cohen-Heist, method, in which a culture from an infected area or discharge is planted simultaneously in a deep tube of rich culture medium, such as dextrose–brain broth, and in an empty sterile test tube into which several cubic centimeters of the patient's fresh, whole, coagulable blood is afterward placed.[9] This method has been used successfully by investigators here and abroad, a number of whom have modified and improved it. Heist and I,[6a] Lowe,[6d] Shearman [6e] and Crowe [6f] have made control experiments proving its reliability. Crowe [6f]

6. (*a*) Solis-Cohen, M., and Heist, G. D.: A Method of Distinguishing from Among Various Microörganisms Present in a Patient, Those That Are and Those That Are Not Acted upon by That Patient's Whole Coagulable Blood, Pennsylvania M. J. **25**:27 (Oct.) 1921. (*b*) Solis-Cohen, M.: Accentuating Pathogenic Organisms in Culture by Utilizing the Inhibitory Influence of Whole Blood, Brit. J. Exper. Path. **8**:149 (June) 1927. (*c*) Boerner, F., and Solis-Cohen, M.: A Study of Pathogen-Selective Cultures in Relation to Vaccine Therapy, Am. J. Clin. Path. **3**:125 (March) 1933. (*d*) Lowe, E. C.: Pathogen-Selective Cultures as an Aid to the Diagnosis of Infective Foci, Brit. M. J. **2**:98 (July 21) 1928. (*e*) Shearman, C. H.: The Application of Pathogen-Selective Methods of Culture in the Investigation of Foci of Infection in Arthritis, M. J. Australia **2**:662 (Nov. 9) 1929. (*f*) Crowe, H. W.: Pathogen-Selective Culture and Its Bearing on the Classification and Etiology of Chronic Rheumatic Disease, in Charterhouse Rheumatism Clinic Original Papers, London, Oxford University Press, 1937, vol. 1, p. 3.

7. Solis-Cohen, M.: (*a*) Determining the Infectivity of Bacteria for Their Host, with Special Reference to the Pathogen-Selective Culture, Lancet **2**:1447 (Dec. 19) 1936; (*b*) Vaccine Therapy in Chronic and Focal Infections, Internat. Clin. **2**:214 (June) 1939.

8. Solis-Cohen and Heist.[6a] Solis-Cohen (footnotes 6 *b* and 7).

9. Kolmer, J. A., and Boerner, F.: Approved Laboratory Technic, New York, D. Appleton and Company, 1931. Footnotes 6 and 7.

stated that when the vaccine treatment of any chronic disease of unknown origin is to be undertaken this test should never be omitted.

The pathogen-selective vaccine consists of 9 parts of a broth culture of the organisms that grew in the patient's blood and 1 part of a broth culture of the organisms that grew in the rich culture medium.[9] This vaccine is extremely potent and in most cases must be given in very small doses. There is no standard dose. One patient may require a dose one trillionth of that needed by another patient.[7b]

Bacterial infection frequently produces hypersensitiveness to the infecting microbe and its toxic products, sometimes to a very pronounced degree, which hypersensitiveness may be further increased by the injection of a dose of vaccine too large for the individual patient. Owing to the minute doses that have to be administered in such cases, some immunologists draw an unjustified analogy from protein desensitization, in which minute doses are also employed, and conclude that the purpose of vaccine therapy is to desensitize the patient. In the opinion of Topley and Wilson,[10] however, the view that an infected animal will benefit from the neutralization of antibody by antigen necessitates a radical alteration in the general conceptions of immunity and has, at the moment, slight basis in experimental fact.

Successful vaccine therapy requires skilful administration, which means giving the dose appropriate to the individual patient at the appropriate interval.[7b] Most physicians begin with a standard dose. I [7b] endeavor to determine beforehand the appropriate initial dose for each patient by injecting intracutaneously in the forearm 0.05 cc. of a control broth and three test doses of 0.05 cc. each, the largest containing from 1 to 50,000,000 organisms, the next containing a tenth to a thousandth of this number and the last a tenth to a thousandth of the second dose. The amounts in the test doses depend on the type, stage, severity and duration of the disease, the degree of toxemia present and the condition of the patient. The smallest amount that gives a slight local reaction is administered therapeutically. If all the test doses cause a pronounced reaction, another series of intracutaneous tests with still smaller amounts is given. Each subsequent dose is regulated in accordance with the general, focal and local reactions produced by the preceding dose.

The appropriate dose of vaccine, in my opinion, is that amount which produces a favorable reaction, an amelioration of the symptoms and the longest period of improvement. I repeat or increase the dose which causes a favorable or very slightly unfavorable general or focal reaction, slight or no local reaction or improvement without reaction. When

10. Topley, W. W. C., and Wilson, G. S.: The Principles of Bacteriology and Immunity, London, Edward Arnold & Company, 1929, vol. 2.

there is a slight to moderate unfavorable general or focal reaction or a moderate local reaction, the dose is reduced to from one half to one thousandth of the amount causing such a reaction. A marked unfavorable general or focal reaction or a decided local reaction is regarded as a warning to reduce the dose to from one-thousandth to one-millionth or one-billionth. The dose is increased when there is no reaction and the patient shows no improvement.

The dose should be given every five days, or preferably just as the favorable effect of the preceding dose is beginning to wear off and the symptoms of the disease show signs of returning or increasing, or just before this occurs. A slight unfavorable reaction is always allowed to subside for a day or two, a moderate unfavorable reaction for three to seven days and a severe unfavorable reaction for ten days to four weeks before the next dose is given. As the patient improves, the interval may be lengthened to one, two or three weeks or months or longer. When no symptoms return, further vaccine treatment is not necessary.

In the treatment of chronic sinusitis, as in that of other diseases, vaccine therapy usually is an adjuvant to other therapeutic measures. Nevertheless, there are some types of the disease in which the use of a vaccine is the best or the only indicated treatment.

A number of patients with chronic sinusitis were stated by Dr. Robert F. Ridpath to be unsuitable at the time for either operative or local treatment, owing to the condition of the mucous membrane. On his recommendation they were given pathogen-selective vaccines and then reexamined by him. At the second examination several patients were found in suitable condition for operation, while others were pronounced cured and in no need of any treatment.

There are cases of chronic sinusitis in which the disease is more or less quiescent when the patient's resistance is high and exacerbations occur when his resistance is temporarily lowered, such exacerbations frequently being regarded as recurrent colds. I have found pathogen-selective vaccines efficacious in such cases of chronic sinusitis.

Many patients suffer from systemic disease or bacterial toxemia, secondary to a latent or purely bacterial sinal infection, unaccompanied by local symptoms or any abnormality that can be detected by an expert rhinologist. Such a condition is common after an attack of grip or pneumonia, and I have referred to it as "lingering influenza."[11] These patients frequently have been cured of their systemic disease and toxemia by the administration of a pathogen-selective vaccine.

Sometimes patients with demonstrable chronic sinusitis and secondary systemic disease and toxemia do not require operation but fail to respond

11. Solis-Cohen, M.: Lingering Influenza, Am. J. Clin. Med. **31**:11 (Jan.) 1924.

to local treatment, even in the most expert hands. Vaccine has cured or relieved many such patients both of their sinusitis and of their systemic disease and toxemia.

In other patients the local symptoms of chronic sinusitis are controlled by regular, continuous local treatment, which, however, has no effect on the systemic disease or the toxemia. These patients need vaccine to increase their ability to manufacture in sufficient quantity the antibodies necessary to cure the systemic disease and the toxemia. It may be necessary with all the types of chronic sinusitis mentioned to maintain drainage by means of shrinking solutions sprayed or dropped in the nose.

Possibly the most unfortunate group of patients with chronic sinusitis are those who have had one or more nasal or sinal operations without obtaining relief either from the sinusitis itself or from the secondary systemic disease or the toxemia. The otolaryngologic journals contain many articles testifying to the frequent failure of radical operations to cure sinusitis.[12] As expressed by Proetz,[13]

> We know . . . that from a shocking percentage of sinus operations no relief is obtained; in fact, the operation often aggravates the symptoms or produces new ones. . . . The sophistry "once a sinus always a sinus," which the laity now sententiously quote, unfortunately . . . comes too near the truth.

In my opinion,[12] the reason for the frequent unsuccessful result of treating chronic sinusitis by radical or conservative measures is the failure to realize that these measures do not eradicate the infection. After operation the infecting organisms often remain in the sinuses, nares and nasopharynx, and sometimes on the tonsils and fauces, where they continue to live and multiply and manufacture toxins.[14] Not until the patient's defensive forces are increased, so that they overcome the infecting bacteria and their toxins, does a cure result. This sometimes occurs spontaneously after the operation, especially when particular attention is paid to hygienic measures. But in most cases it is necessary in addition to give a vaccine in order to stimulate artificially the production of antibodies to destroy the infecting microbes and render their toxic products harmless.

The trauma produced by a perfect operation may so reduce both local and general resistance that healing is retarded. In addition, it sometimes enables the infecting bacteria to enter the blood stream, where

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12. Solis-Cohen, M.: Why Otolaryngologists Frequently Fail to Remove a Focus of Infection, Ann. Otol., Rhin. & Laryng. **43**:586 (June) 1934.

13. Proetz, A. W.: The Sinus in Perspective, Ann. Otol., Rhin. & Laryng. **38**:682 (Sept.) 1929.

14. Solis-Cohen, M.: Necessity for Revising the Common Conception of Focal Infection, J. A. M. A. **102**:1128 (April 7) 1934.

they may live for a long time and produce septicemia. Or they may survive for only a brief period, but long enough to reach tissue suitable for their propagation and growth, where they colonize and metastasize, forming a secondary focus of infection. Even a slight operation has been followed by exacerbation of symptoms, and even death. These dangers may sometimes be averted by raising resistance to the infecting microbe prior to operation through the administration of a vaccine. This is particularly necessary in cases of rheumatism and heart disease.

When sinusitis develops in allergic persons, the secondary effects frequently take the form of bacterial allergy, with asthma and allergic rhinitis as the prominent manifestations. This is seen in cases of so-called perennial hay fever, in which the chief offending substances are not pollens but bacterial toxins. Another example frequently encountered is the child who has asthma with every infection of the upper respiratory tract, which in many instances is an acute exacerbation of chronic sinusitis. Vaccine therapy is extremely helpful in such cases. With the cure of the bacterial infection, the asthmatic attacks frequently disappear and the hay fever may be either completely cured or decidedly improved and rendered amenable to pollen desensitization, which before had been unsuccessful.

Vaccine therapy may be of no benefit to persons with chronic sinusitis when there is suppuration in a sinus that is not draining or that drains but poorly and when the sinusitis is secondary to an untreated primary focus of infection, such as diseased tonsils. It may even increase the symptoms under these conditions, especially when hypersensitivity is present and the dose is large.

CONCLUSIONS

Chronic sinusitis and the systemic disease and bacterial toxemia secondary to it will be cured more frequently when physicians recognize that the surgeon's knife may remove infected tissue and promote drainage but does not eradicate infection, that infection can exist in a sinus in the absence of visual evidence of disease and that infection is cured only when the body manufactures sufficient antibodies to combat the infecting organism and its toxic products. As a means of stimulating such antibody production the administration of a potent vaccine, containing the etiologic organism, is frequently necessary.

2110 Spruce Street.

OTITIS MEDIA AND MASTOIDITIS IN CHILDREN

BACTERIOLOGIC STUDIES WITH SPECIAL REFERENCE TO THE PATHOGENICITY OF STAPHYLOCOCCI

E. NETER, M.D.

F. NEUBURGER, M.D.

AND

M. L. SHORT, A.B.

BUFFALO

The most important micro-organisms responsible for otitis media and mastoiditis are group A hemolytic streptococci, pneumococci and staphylococci. There is, however, a marked difference in incidence of these bacteria between otitis media and mastoiditis, inasmuch as staphylococci are rather frequently encountered in the former condition but only rarely in the latter. The question arises, therefore, whether or not staphylococci are the cause of otitis media without tendency to invade the mastoid cells or whether staphylococci found in otitis media may be nonpathogenic and outgrow on culture the true pathogens. It is generally agreed today that the best in vitro method to determine pathogenicity or nonpathogenicity of staphylococci is the plasma coagulation test. Pigment and hemolysin production, formerly considered to reflect pathogenicity, are not as reliable indicators as the production of coagulase. It was decided, therefore, to carry out a bacteriologic study on cases of otitis media and mastoiditis and to determine the pathogenicity of the staphylococci, in order to throw further light on their etiologic significance in these conditions.

MATERIAL AND METHODS

The material is comprised only of cases that were studied bacteriologically from January 1939 to May 1941. All cases of mastoiditis and the majority of the cases of otitis media were those of patients admitted to the Children's Hospital; only a few cases of otitis media were included from the outpatient clinic. Many cases of otitis media with treatment at this hospital were not studied bacteriologicaliy. These facts account for the relatively large number of cases of mastoiditis and the relatively small number of those of otitis media. Furthermore, it may be mentioned that in many cases of mastoiditis cultures of material from the middle ear were not made.

From the Departments of Bacteriology and Otolaryngology of the Children's Hospital and the University of Buffalo, School of Medicine.

The material for culture in cases of otitis media was taken by means of a swab after careful cleaning of the aural canal. If it was not possible to seed the culture mediums immediately after taking the material, Brahdy swabs containing coagulated serum as a culture medium were used. These swabs gave very satisfactory results with outpatients. A swab was put into infusion broth and streaked on blood agar plates. The latter were prepared with human blood. When indicated, special mediums, such as chocolate agar for Bacillus influenzae or liver broth for anaerobic bacteria, were added. The types of Pneumococcus were determined by agglutination and precipitation tests. The antipneumococcus horse serums (types I to XXXII) were obtained from the New York City Department of Health, through the courtesy of Dr. Ralph S. Muckenfuss. It may be mentioned in this connection that the presence of blood in the broth may interfere with agglutination and precipitation. In instances of this kind typing had to be carried

TABLE 1.—*Bacteriologic Observations in Cases of Mastoiditis and Otitis Media in Children*

Micro-Organisms	Mastoiditis		Otitis Media	
	Cases	Approximate Percentage	Cases	Approximate Percentage
Streptococcus haemolyticus	80	75	58	13.5
Streptococcus haemolyticus and staphylococcus	5	5	63	14.5
Streptococcus haemolyticus and other bacteria	0	0	10	2.5
Pneumococcus	12	11	85	18.5
Pneumococcus and staphylococcus	2	2	15	3.5
Staphylococcus	1	1	158	36
Bacillus influenzae (alone or with staphylococcus)	0	0	5	1.25
Bacillus proteus (overgrowing)	2	2	9	2
Friedländer's bacillus	0	0	6	1.5
Bacillus pyocyaneus (alone or overgrowing)	0	0	3	0.75
Bacillus alcaligenes	0	0	1	0.25
Micrococcus catarrhalis and staphylococcus	0	0	2	0.5
Micrococcus tetragenus and staphylococcus	0	0	1	0.25
Streptococcus viridans	0	0	1	0.25
Bacillus subtilis	2	2	9	2
Diphtheroid bacilli	1	1	3	0.75
Bacillus funduliformis	1	1	0	0
No growth	0	0	9	2
Total	106	100	438	100

out on subcultures. In some cases the material was injected into mice, if attempts to isolate the pneumococci by other methods failed.

The isolated strains of staphylococci were tested for their capacity to produce hemolysin, pigment and plasma coagulase. In order to demonstrate plasma coagulase the method recently recommended by Fisk[1] was employed.

RESULTS

A bacteriologic study was carried out in 106 cases of mastoiditis and 438 cases of purulent otitis media in children. The results of this investigation are summarized in table 1.

The table shows that hemolytic streptococci were found in approximately 80 per cent of 106 cases of mastoiditis and pneumococci in 13 per

1. Fisk, A.: The Technique of the Coagulase Test for Staphylococci, Brit. J. Exper. Path. **21**:311-314 (Oct.) 1940.

cent. It is interesting to note that in the majority of cases hemolytic streptococci and pneumococci were encountered in pure culture and that in only a small fraction of the cases (5 per cent and 2 per cent, respectively) were these organisms associated with staphylococci. In this series there was only 1 case in which staphylococci were found as the sole infection. Bacillus proteus occurred in 2 and the anaerobic Bacillus funduliformis in 1 of the cases. In 2 cases Bacillus subtilis and in 1 diphtheroid bacilli were recovered; these organisms are to be considered as contaminants. It may be emphasized that in this series no cultures from patients with mastoiditis were seen which failed to show growth.

TABLE 2.—*Incidence of the Different Types of Pneumococci in Mastoiditis and Otitis Media of Children*

Type of Pneumococcus	Cases of Mastoiditis	Cases of Otitis Media
I	..	11
II	..	3
III	5	7
IV	..	7
V	..	4
VII	4	21
VIII	..	7
IX	..	2
XI	..	1
XII	..	1
XIV	2	10
XVI	1	1
XVIII	..	2
XIX	1	10
XX	..	6
XXI	..	2
XXII	1	0
XXIX	..	1
Unclassified	..	4
Total	14	100

The bacteriologic findings in 438 cases of purulent otitis media differ in certain respects from those obtained in mastoiditis. The incidence of hemolytic streptococci is only 30.5 per cent, as contrasted with 80 per cent in cases of mastoiditis. Pneumococci were found in 22 per cent of the cases of otitis media. Staphylococci were encountered rather frequently in cases of otitis media and accounted for the disease in 36 per cent of the cases, while this organism was found in only 1 per cent of the cases of mastoiditis.

Table 1 shows, furthermore, that the number of mixed infections (hemolytic streptococcus and staphylococcus; pneumococcus and staphylococcus) is definitely greater for cases of otitis media than for cases of mastoiditis.

The incidence of the different types of pneumococci in cases of mastoiditis and of otitis media is summarized in table 2. It is interesting

to note that types I and II were not encountered in 14 cases of mastoiditis but were encountered in 14 of 100 cases of otitis media. In contrast, type III was found in 5 of 14 cases of mastoiditis but in only 7 of 100 cases of otitis media. Of the higher types of pneumococci, types VII, XIV and XIX occurred rather frequently.

In view of the fact that staphylococci in pure culture were recovered in 158 (36 per cent) of 438 cases of otitis media in children, the question of the pathogenic significance of this organism in otitis media presents itself. It is possible that staphylococci on culture outgrew the true pathogen and were more or less nonpathogenic secondary invaders. In favor of this argument it may be pointed out that in this series at least staphylococci only very rarely occurred in cases of mastoiditis. On the other hand, it is possible that staphylococci are pathogenic and produce inflammatory lesions of the middle ear but that they do not have the tendency to spread further and thus cause mastoiditis. In order to elucidate this matter, studies were carried out to determine the pathogenicity of staphylococci which were recovered in cases of otitis media and mastoiditis. It is generally recognized (Pijoan,[2] Fisher,[3] Cruickshank,[4] Luecking,[5] Blair,[6] Fairbrother,[7] Fisk,[1] Chapman, Berens and Stiles [8] and others) that of all in vitro tests available the plasma coagulase test is the most reliable for this purpose. Moreover, the plasma-clotting factor of staphylococci has been demonstrated in vivo (Neter [9]).

The plasma-clotting properties of 32 strains of staphylococci isolated from patients with otitis media and mastoiditis were studied. It was found that 19 strains produced coagulase, while 13 strains failed to do so. Although the majority of these 32 strains produced both pigment and hemolysin, an occasional strain of nonhemolytic staphylococci

2. Pijoan, M.: A Study on the Blood-Coagulating Substance Produced by Staphylococci and Its Relation to Disease, Canad. M. A. J. **32**:476-481 (May) 1935.

3. Fisher, A. M.: The Plasma-Coagulating Properties of Staphylococci, Bull. Johns Hopkins Hosp. **59**:393-414 (Dec.) 1936.

4. Cruickshank, R.: Staphylocoagulase, J. Path. & Bact. **45**:295-303 (July) 1937.

5. Luecking, B.: Ueber die Methoden zur Bestimmung der Pathogenität der Staphylokokken unter besonderer Berücksichtigung der Blutplasmakoagulation, Inaug. Dissert., Bonn, Feb. 17, 1937.

6. Blair, J. E.: The Pathogenic Staphylococci, Bact. Rev. **3**:97-146 (June) 1939.

7. Fairbrother, R. W.: Coagulase Production as a Criterion for the Classification of the Staphylococci, J. Path. & Bact. **50**:83-88 (Jan.) 1940.

8. Chapman, G. H.; Berens, C., and Stiles, M. H.: The Coagulation of Plasma by Staphylococci, J. Bact. **41**:431-440 (April) 1941.

9. Neter, E.: Fibrinolytic, Anticoagulating and Plasma-Clotting Properties of Staphylococci, J. Bact. **34**:243-254 (Sept.) 1937.

produced coagulase. It is interesting to note that repeated examinations of the strains gave identical results. In parallel tests it was found that broth cultures gave more satisfactory results than suspensions prepared from plain agar plates. The tests, furthermore, indicated that young broth cultures (three to six hours) are suitable only if sufficient growth has taken place during this short period of incubation; otherwise, falsely negative results may be obtained. It may be important to emphasize that adequate control studies with uninoculated broth should always be carried out, since occasionally spontaneous coagulation may occur. In our hands eighteen to twenty-four hour infusion broth cultures diluted 1:10 in physiologic solution of sodium chloride gave satisfactory results. The investigation, furthermore, revealed that both coagulase-positive and coagulase-negative strains may be obtained in pure culture or in association with pathogens such as the hemolytic streptococcus and the pneumococcus. Two of the strains tested were recovered from patients with mastoiditis; both strains failed to produce coagulase. In a few cases of acute suppurative otitis media material for culture was taken simultaneously from the aural canal and the middle ear. In some of these cases coagulase-negative strains and in others coagulase-positive strains were obtained from both sources. Finally, it may be mentioned that in a few instances two different strains of staphylococci were obtained from the middle ear in cases of otitis media—one coagulase-positive, the other coagulase-negative. It may be concluded from these observations that certain strains of staphylococci recovered from the lesions of purulent otitis media and mastoiditis are not pathogenic and therefore can be excluded as causative in these instances. On the other hand, strains of staphylococci pathogenic for man may be present in these lesions.

COMMENT

The bacteriologic investigation of otitis media and mastoiditis resulted in several interesting findings. In the first place, it could be shown that carefully made cultures in cases of mastoiditis give satisfactory results: In the majority of cases (93 per cent) hemolytic streptococci and pneumococci, which are generally recognized as true pathogens responsible for this condition, were encountered; the former accounted for the disease in 80 per cent and the latter in 13 per cent of all cases. In the remaining 7 per cent of these cases staphylococci were found in only 1 instance, B. proteus in 2, B. funduliformis and diphtheroid bacilli in 1 each and B. subtilis in 2 instances. It is noteworthy, furthermore, that in only 7 per cent of all cases were true pathogens (hemolytic streptococci and pneumococci) associated with staphylococci. It may be mentioned in this connection that B. proteus not only may be a

relatively harmless secondary invader in otitis media and mastoiditis but may occasionally cause fatal bacteremia and meningitis (Neter and Chait [10]).

Bowers [11] recently reported on the bacteriologic findings in cases of mastoiditis. He found staphylococci to be present in no less than 15 per cent. He raised the question as to the possibility either that the staphylococci were contaminants or that these organisms outgrew the more serious bacteria, such as hemolytic streptococci and pneumococci. Our series seems to support this assumption of Bowers, indicating that staphylococci play only a minor role in acute mastoiditis of children.

Bowers reported that 18 per cent of cultures of material taken at operation in cases of mastoiditis failed to reveal growth. He considered it probable that in the greater part of these cases the mastoiditis was caused by streptococci and pneumococci. In our series of 106 cases of mastoiditis there were only 3 in which nonpathogenic organisms (B. subtilis and diphtheroid bacilli) were recovered, and none in which a culture failed to reveal growth. Thus, our findings substantiate the assumption of Bowers [11] and clearly show that cultures of material taken at operations for acute suppurative mastoiditis in children give very satisfactory results in the majority of cases and, furthermore, that nonpathogenic organisms or sterile cultures are obtained only in a small percentage of cases.

Another finding deserves a brief discussion. It has been pointed out in the literature that nonhemolytic streptococci may cause mastoiditis. Although this cannot be denied, it seems unlikely that such an occurrence is relatively frequent. Neither nonhemolytic streptococci nor Streptococcus viridans was encountered in our series of 106 cases of mastoiditis. It may be mentioned in this connection that some strains of hemolytic streptococci on first isolation may fail to produce hemolysis on blood agar plates. Repeated subcultures on blood agar plates, the demonstration of hemolysin in fluid mediums or the employment of horse blood instead of human or rabbit blood may give evidence that the isolated organisms should be classified as hemolytic streptococci. It is suggested, therefore, that these methods be employed in all cases of mastoiditis in which the "nonhemolytic" streptococcus or Str. viridans is first isolated.

The bacteriologic findings in purulent otitis media differ markedly from those obtained in acute mastoiditis. The most striking difference is in the incidence of staphylococci. In our study staphylococci were

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10. Neter, E., and Chait, R. A.: Bacillus Proteus in Otitis Media, Complicated by Mastoiditis, Meningitis and Bacteremia, Arch. Otolaryng. **32**:947-950 (Nov.) 1940.

11. Bowers, W. C.: Observations on Seven Hundred and Ninety-Three Cases of Acute Purulent Otitis Media, with Chemotherapy in Three Hundred and Ninety-Six Cases, J. A. M. A. **115**:178-181 (July 20) 1940.

found in no less than 36 per cent of 438 cases in pure culture and in an additional 18 per cent in association with hemolytic streptococci or pneumococci. This total of 54 per cent contrasts with an incidence of only 8 per cent in mastoiditis. This observation prompted an in vitro study on the pathogenicity of staphylococci encountered in otitis media. The results will be discussed in a subsequent paragraph.

The reports on the incidence of staphylococci in cases of otitis media show marked discrepancies. Henry and Kuhn [12] found staphylococci in 113 of 489 instances (24 per cent); an additional 17 per cent of these cases presented staphylococci in association with other organisms. In contrast, Stuart [13] found Staphylococcus pyogenes in only 40 of 754 cases of acute suppurative otitis media. This author, however, did not indicate whether nonpathogenic staphylococci were excluded from his series.

A comparative study on the incidence of the different types of pneumococci in cases of otitis media and of mastoiditis revealed several interesting facts. In the first place, it was confirmed that in mastoiditis type III pneumococci are the most important of the lower types of pneumococci; in our series, this type was encountered in 5 of 14 cases. On the other hand, type III pneumococci were recovered in only 7 of 100 cases of otitis media. It is interesting to note that type I pneumococci were found in 11 of 100 cases of otitis media, whereas this type was not encountered in any of our 14 cases of pneumococcic mastoiditis. This does not indicate that type I pneumococci could not cause mastoiditis. However, these observations suggest that the invasiveness of bacteria may depend not only on properties of particular bacterial species but also on those of different types and individual strains.

Many of the higher types (types IV to XXXIII) of pneumococci were encountered in otitis media; some, such as types VII, XIV and XIX, were seen more frequently than others. There can be little doubt that there are variations in the incidence of various types of pneumococci at different times and in different locations. It is therefore not surprising to see that marked discrepancies exist in the reports of various authors in regard to the incidence of various types of pneumococci in otitis media and mastoiditis. Stuart [13] found pneumococci of type III in 71 of 754 cases of acute otitis media and pneumococci of other types in 67. Of our series of 100 cases of pneumococcic otitis media, pneumococci of type III accounted for only 7 per cent. Eighteen different types

12. Henry, L. D., and Kuhn, H. A.: Bacteriological Studies of Acute Infections of the Middle Ear, Ann. Otol., Rhin. & Laryng. **49**:519-525 (June) 1940.

13. Stuart, E. A.: Acute Suppurative Otitis Media: A Review of Cases Encountered During a Five Year Period, Arch. Otolaryng. **33**:734-748 (May) 1941.

of pneumococci were found in our series, presenting evidence that, in children at least, higher types of pneumococci may play an important role in this condition.

Bowers [11] encountered pneumococci of "Group 3" in 5 per cent of his cases of mastoiditis and did not recover any of the other types of pneumococci. Our series indicates that lower and higher types of pneumococci may cause mastoiditis, although, of the lower types, type III seems to be particularly apt to do so.

The in vitro studies on the pathogenicity of staphylococci recovered from lesions in otitis media and mastoiditis here reported revealed that both pathogenic and nonpathogenic strains may be present. In a series of 32 cases, pathogenic strains were isolated in 19 and nonpathogenic strains in 13. In some of these cases nonpathogenic staphylococci were found in pure culture. It is evident, therefore, that in these instances the causative agent was not recovered and that the bacteriologic findings failed to explain the nature of the infection. It is recommended that the search for other organisms in these instances be continued, either by employing more delicate bacteriologic methods or by making repeated cultures. Injection of suspected material into mice may reveal the presence of pneumococci when ordinary cultures fail, and the addition of polyvalent staphylococcus phage to the culture medium may possibly help in the isolation of the true pathogen. On the other hand, the isolation of pathogenic staphylococci does not necessarily indicate that these organisms are the primary causative agent or, as secondary invaders, responsible for the continuation of the infection. Their role in the pathogenesis of otitis media needs further elucidation. On the basis of our observations, the routine use of the plasma coagulase test as an aid in the interpretation of bacteriologic findings in otitis media and mastoiditis is highly recommended.

SUMMARY

A bacteriologic study of 106 cases of mastoiditis and 438 cases of otitis media with particular reference to the pathogenicity of staphylococci yielded the following data:

Acute Mastoiditis (106 cases).—1. Hemolytic streptococci were found in 75 per cent of the cases in pure culture and in 5 per cent together with staphylococci.

2. Pneumococci were recovered in 11 per cent in pure culture and in 2 per cent together with staphylococci. The incidence of the different types is presented.

3. Staphylococci in pure culture accounted for only 1 per cent of the cases.

4. Sterile cultures or contaminants were obtained in a small percentage of the cases only (3 per cent).

Otitis Media (438 cases).—1. Hemolytic streptococci were found in pure culture in 13.5 per cent and together with other bacteria in 17 per cent.

2. Pneumococci were recovered in pure culture in 18.5 per cent and associated with staphylococci in 3.5 per cent. The incidence of the different types of pneumococci is given.

3. Staphylococci in pure culture were recovered in no less than 36 per cent of the cases.

The incidence of other micro-organisms is presented.

Pathogenicity of the Isolated Strains of Staphylococci.—Thirteen of 32 strains of staphylococci isolated from lesions of otitis media and mastoiditis were found to be nonpathogenic according to the plasma coagulase test. The finding of nonpathogenic staphylococci in pure culture in some cases indicates that the causative agent was not determined. Pathogenic staphylococci isolated from otitis media were encountered either in pure culture or in association with other pathogens.

CONCLUSIONS

Cultures of material from lesions of acute suppurative mastoiditis in children reveal true pathogens in the vast majority of cases. Staphylococci play only a minor role.

The routine use of the plasma coagulase test is recommended as an aid in the interpretation of the significance of staphylococci isolated from otitis media and mastoiditis. The employment of additional bacteriologic tests in cases in which only nonpathogenic staphylococci are encountered may further increase the percentage of satisfactory bacteriologic examinations.

219 Bryant Street.

Case Reports

CHONDROMA OF THE LARYNX

PAUL H. HOLINGER, M.D., AND LLOYD L. MATZKIN, M.D., CHICAGO

A chondroma is a relatively rare tumor of the larynx. It is of interest because its insidious onset frequently allows the patient to procrastinate regarding treatment until radical resection may be necessary to eliminate the simple, benign tumor.

REPORT OF A CASE

History.—A. W., a 40 year old French-Irish bricklayer, entered the Research and Educational Hospital on Feb. 7, 1941, complaining of hoarseness and a change in the quality of his voice of eighteen months' duration. He stated he had been aware of a swelling on the left side of his neck, which he first noticed about two years before admission, but it was not until about six months later that he associated the mass with his gradually increasing hoarseness. He was aware that in certain positions of his head and neck, such as hyperextension, or when he attempted to reach high tones while singing, his hoarseness became severe and was associated with a sense of pressure or of a foreign body in the throat. The hoarseness and swelling progressed slowly, and finally the sensation of a foreign body in his throat became constant and caused him to cough and clear his throat continually in an attempt to relieve this sensation. Prior to the present illness he had been entirely well.

Examination.—Physical examination revealed a muscular, healthy man who had no apparent defects except the tumor in the neck. Here, attached to the left thyroid plate, could be felt a hard, globular mass, 3 by 2 cm., lying under a freely movable skin, moving with the thyroid cartilage on swallowing and apparently forming an integral part of it. The mass was well defined and appeared to have a smooth surface. There was no associated cervical lymphadenopathy. Mirror laryngoscopy showed a smooth, rounded supraglottic swelling extending more than halfway into the lumen of the glottis from the left side and covered by a thickened, moderately inflamed mucous membrane. The left false vocal cord, arytenoid cartilage and aryepiglottic fold were involved in the tumor. The left true vocal cord could not be visualized because of the overlying mass. The right cord was entirely free and exhibited good motion. Because of the intralaryngeal swelling the rima glottidis was greatly narrowed but still remained patent enough to provide a fair-sized airway. Direct laryngoscopy did not add to the information obtained by the mirror examination except to indicate that in its intralaryngeal extent the tumor was of the same consistency as noted by external palpation. The tumor could not be reflected to allow examination of the true cord.

Roentgen examinations, both by flat plates and by planograms, gave excellent evidence of the extent of the tumor, confirming the observations as to the size, shape and consistency of the mass already described.

From the Department of Laryngology, Rhinology and Otology, the University of Illinois College of Medicine.

Fig. 1.—Chondroma arising from the left wing of the thyroid cartilage (mirror view of the larynx).

Fig. 2.—Lateral roentgenogram of the soft tissues of the neck. The laryngeal ventricle is visible and does not appear to be distorted. The tumor may be outlined above the ventricle.

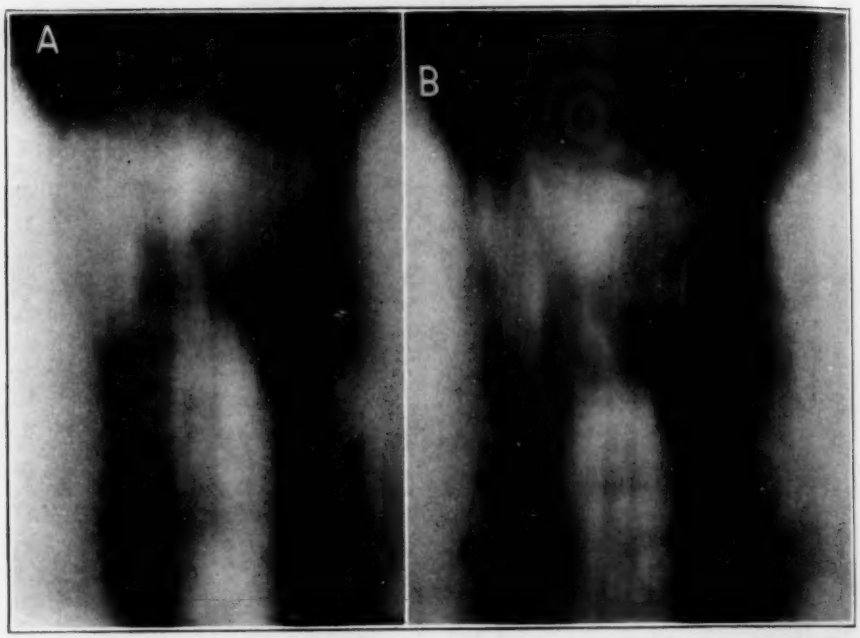

Fig. 3.—Planograms of the larynx (*A*) at the 1 cm. level and (*B*) at the 2 cm. level. In *B* the hyoid bone is clearly distinguishable. The tumor is seen encroaching on the laryngeal lumen.

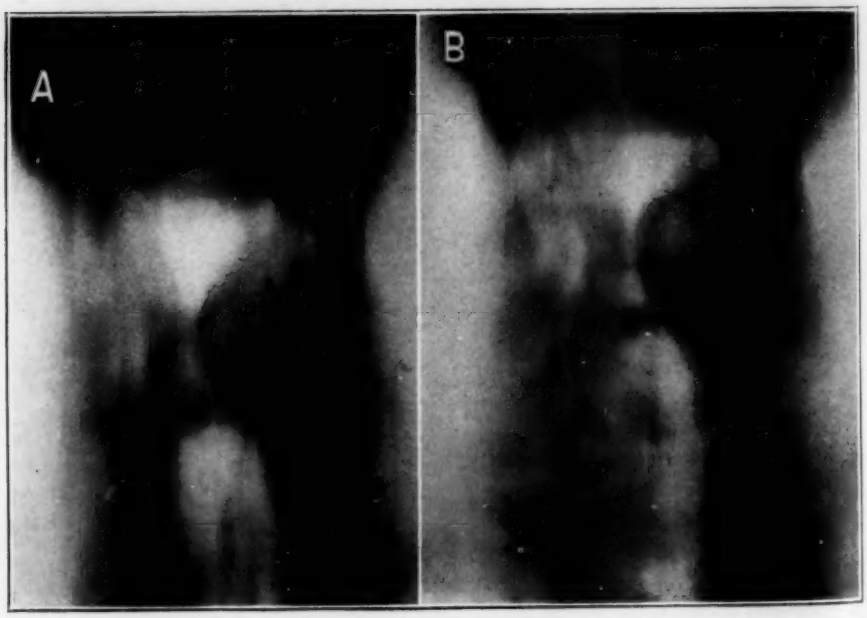

Fig. 4.—Planograms of the larynx (*A*) at the 4 cm. level and (*B*) at the 6 cm. level.

Diagnosis.—A tentative diagnosis of chondroma of the larynx was made and surgical removal elected.

Operation.—On February 18, the laryngeal mucosa having been sprayed with a 2 per cent solution of pontocaine hydrochloride and anesthesia induced by endotracheal administration of cyclopropane and ether, a left horizontal incision was made in the neck, at the level of the middle of the thyroid cartilage, directly overlying the mass. The skin was reflected and a vertical separation made in the

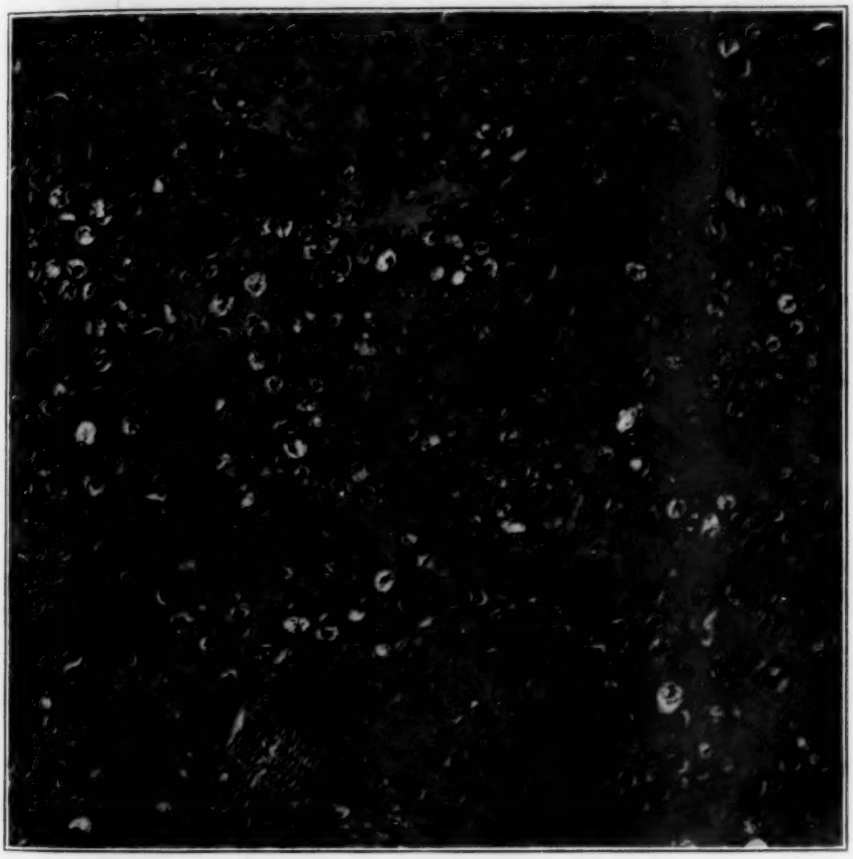

Fig. 5.—Photomicrograph of a section of the tumor, showing the typical structure of the chondroma (× 150).

body of the platysma muscle to expose the tumor immediately. The growth was observed to involve the entire left lateral plate of the thyroid cartilage. The surface of the tumor was hard and round and had a "pebble-grained" finish. An attempt was then made entirely to free the tumor through a submucous separation of the tumor from the laryngeal mucous membrane and evert it from its hard, cartilaginous bed by means of blunt dissection. Because of its friable nature, however, it could not be shelled out in one piece and had to be removed by morsella-

tion and curettement. The laryngeal mucous membrane was left intact. An iodoform gauze drain was inserted into the operative defect and the external wound closed with catgut and silk. The patient made a satisfactory recovery except for moderate laryngeal edema on the second postoperative day and was discharged from the hospital February 25, on the seventh postoperative day.

The specimen consisted of several small and one large piece of uniformly grayish, friable, cartilaginous material, which cut with some resistance and when

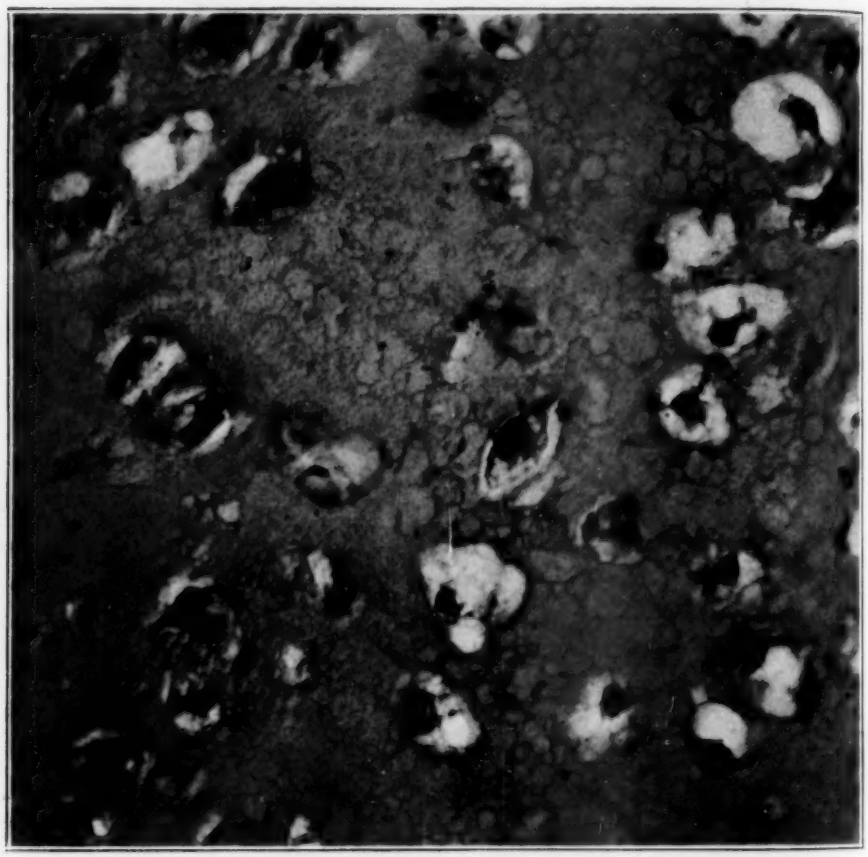

Fig. 6.—Appearance of the tumor under high power (× 550).

reconstructed measured approximately 3 by 3 by 2.5 cm. On microscopic examination the tissue was seen to be composed of hyaline cartilage, showing areas of degeneration and several large vascular sinuses. The clinical diagnosis of a benign chondroma of the thyroid cartilage was confirmed by the histologic study of the tissue removed at operation.

One month after discharge from the hospital the patient was again observed. The external wound had completely healed. Mirror laryngoscopy revealed an entirely normal picture. The right and left true cords moved freely; the supraglottic swelling had disappeared, and the patient had a normal speaking voice.

COMMENT

The early literature was thoroughly studied and a series of cases of chondroma of the larynx analyzed by New [1] and Moore.[2] Moore collected 62 cases from the literature, including his own, up to 1925 and concluded that laryngeal chondroma is a solitary, immovable, slow growing neoplasm, having either benign or, infrequently, mildly malignant characteristics. He stated that the tumor varied in size from that of a cherry stone to larynx-filling proportions and was generally covered by a normal or only slightly hyperemic mucous membrane. The most frequent site of origin in Moore's series was the endolaryngeal surface of the posterior plate of the cricoid cartilage; the tumor was more common in males than in females, and the highest incidence was in the fifth decade of life. The symptoms noted in other reported cases, namely, hoarseness, dyspnea, cough, aphasia and dysphagia, were all mentioned by the patient whose case is reported here. A review of the literature reveals agreement that this tumor responds solely to surgical extirpation.

Clerf,[3] Figi,[4] Graham,[5] Hennessy [6] and the Jacksons [7] have observed and reported cases, and because of their descriptions one is better able to recognize a condition which, insidious in its onset and course, frequently is seen by the laryngologist so late as to require a major operation for its eradication, when a relatively simple procedure, carried out early, would have sufficed. This is clearly demonstrated by the case reported by Equen and Neuffer,[8] in which a total laryngectomy was required to eliminate the growth. In this regard, the case here presented is of interest because of the complete restoration of function following the relatively early removal of the tumor.

700 North Michigan Avenue.

1. New, G. B.: Cartilaginous Tumors of the Larynx, Laryngoscope **28**:367-376 (May) 1918.

2. Moore, I.: Cartilaginous Tumors of the Larynx: A Study of All Recorded Cases, J. Laryng. & Otol. **40**:9-14 (Jan.); 84-109 (Feb.); 145-164 (March) 1925.

3. Clerf, L. H.: Chondroma of the Larynx, Tr. Am. Laryng. A. **51**:42-51, 1929.

4. Figi, F.: Chondroma of the Larynx, Ann. Otol., Rhin. & Laryng. **41**:369-386 (June) 1932.

5. Graham, H. B.: Chondroma of the Larynx, Ann. Otol., Rhin. & Laryng. **43**:299-303 (March) 1934.

6. Hennessy, R.: Chondroma of the Larynx, J. Laryng. & Otol. **50**:688-689 (Sept.) 1935.

7. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939.

8. Equen, M., and Neuffer, F.: Chondroma Requiring Laryngectomy, South. M. J. **33**:274-279 (March) 1940.

Clinical Notes; New Instruments and Technics

TREATMENT OF BLOCKAGE OF EUSTACHIAN TUBE BY INFLATION WITH AMPHETAMINE VAPOR

LOUIS D. SULMAN, M.D., PHILADELPHIA

Keeping the eustachian tube open is recognized as a necessity in cases of middle ear disease, both to ease pain in acute conditions, such as otitis media, and to prevent deafness resulting from chronic conditions, such as catarrh. With the air passage closed the air present in the middle ear is absorbed and the tympanic membrane is drawn in by the negative pressure thus created. The ossicles become fixed and deafness ensues. Van Dishoeck [1] found that when there was catarrh in the eustachian tube the negative tension of the tympanic membrane usually varied from —10 to —20 cm. of water, dependent on the localization of the disease and the width of the tube. Any infection of the nose and pharynx is apt to clog the eustachian tube. However, the ostium is somewhat protected by folds of cartilage in adults, as shown by Yates [2] in his studies on movement of cilia. The flow is divided into two streams by folds. The larger stream flows anteriorly and the smaller posteriorly, but when the sinuses contain pus divergence of the streams is not complete and the ostium is less protected. Sinusitis may thus be considered a cause of eustachian salpingitis. The protection is even less adequate in the case of children, in whom the lumen is relatively wider and more nearly horizontal and is located lower in relation to the hard palate. At any age lymphoid tissue may obstruct the ostium of the eustachian tube or may change the course of the streams so that they cross the ostium and thus increase the chances of infection of the eustachian tube. Such masses of lymphoid tissue should be removed or irradiated as a preliminary step in treatment of blockage of the eustachian tube.

Valsalva's method of autoinflation of the eustachian tube and forced politzerization with air sometimes cause congestion. Bougienage may cause vasodilatation and destruction of the mucous membrane, for which reason it is rapidly losing favor. Since the lining of the eustachian tube is continuous with that of the nose and is of the same type, it is reasonable to assume that its cilia respond similarly. Astringents in oil impede the action of the cilia and have no effect on the mucus beneath, according to the observations of Proetz.[3]

More than fifty years ago, Politzer [4] advised against the use of fluids in the eustachian tube unless the tympanic membrane was punctured so that the fluids

1. Van Dishoeck, H. A. E.: Measurement of the Tension of the Tympanic Membrane and of the Resistance of the Eustachian Tube, Arch. Otolaryng. **34**: 596-602 (Sept.) 1941.

2. Yates, A. L.: Methods of Estimating the Activity of the Ciliary Epithelium Within the Sinuses, J. Laryng. & Otol. **39**:554-560, 1924.

3. Proetz, A. W.: The Effects of Certain Drugs upon Living Nasal Ciliated Epithelium, Ann. Otol., Rhin. & Laryng. **43**:450-463, 1934.

4. Politzer, A.: A Textbook of Diseases of the Ear, translated and edited by J. P. Cassells, Philadelphia, H. C. Lea's Son, 1883, p. 135.

could escape. He recommended the use of vapors, which he said had previously been used more frequently. The vapors available at that time were steam and the vapors of ammonia, ammonium chloride, chloroform, sulfuric ethers, various ethereal oils, acetic ether, turpentine, iodine and carbon dioxide. Anesthetics and astringents have been found to inhibit ciliary motion,[5] but amphetamine (benzedrine) vapor can be used with impunity. Proetz[5b] reported that it caused no appreciable change in amplitude or rate of the beat during the observation period of twenty minutes. The use of amphetamine to shrink the mucosa of the eustachian tube is logical because this membrane, like the nasal mucosa, has sympathetic innervation. Every rhinologist knows that amphetamine vapor is a very efficient vasoconstrictor.

Wood[6] described a method for insufflating amphetamine vapor directly into the eustachian tube by means of adaptors on the inhaler which allow it to fit into the eustachian catheter. Hulka[7] observed that insufflation of amphetamine vapor into the middle ear relieved tinnitus and did not produce irritation.

To introduce amphetamine vapor into the eustachian tube, shrinkage of the nasal mucosa is effected by the insertion of cotton saturated with a 2 to 3 per cent solution of paredrine hydrobromide (p-hydroxy-α-methylphenethylamine hydrobromide) into the nasal cavity, where it is allowed to remain for ten minutes. The paredrine also shrinks the mucosa at the ostium. The floor of the nose may be treated with a 2 to 3 per cent solution of cocaine in sensitive persons. A eustachian catheter, opening down, is passed along the floor of the nose with gentle rotating motion to avoid any spurs or other obstructions which might be present. When the catheter strikes the posterior pharyngeal wall, it is rotated so that the opening points up and outward. As the catheter is slowly withdrawn it will pass over the fossa of Rosenmüller and strike the cushion formed by the termination of the cartilage of the eustachian tube. After passing the cushion, the catheter will slide into the ostium. With the auscultation tube at the patient's meatus to detect pressure on the tympanic membrane, air is briefly injected into the catheter by means of a Politzer bag. The tympanic membrane, which was previously drawn in, will attempt to return to its normal position during each inflation. This serves to check the position of the catheter. The amphetamine inhaler, fitted with adaptors supplied by the manufacturer, is then inserted in the catheter and attached to the compressed air apparatus. One to three short applications of amphetamine vapor under low regulated pressure are given.

In cases in which introduction of a catheter is too painful, the inhaler with adaptors is placed between the Politzer bag and the nose piece (figure). The nose piece is inserted in one nostril and the other is closed. While the patient puffs out his cheeks, swallows or says "K," amphetamine vapor is propelled into the cavity of the middle ear by pressing the Politzer bag. This method furnishes quick relief and should be used when it is impossible or too unpleasant to pass

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5. (*a*) Lucas, A. M.: The Nasal Cavity and Direction of Fluid by Ciliary Movement in Macacus Rhesus, Am. J. Anat. **50**:141-177, 1932. (*b*) Proetz, A. W.: Further Experiments in the Action of Drugs on Nasal Mucosa, Arch. Otolaryng. **30**:509-515 (Oct.) 1939.

6. Wood, E. L.: A New Drug for Treatment of the Eustachian Tube and Middle Ear, with an Apparatus for Its Use, Arch. Otolaryng. **21**:588-590 (May) 1935.

7. Hulka, J. H.: A Simple Method of Introducing Benzedrine Vapors into the Eustachian Tube, Arch. Otolaryng. **23**:692 (June) 1936.

a catheter. It also eliminates the danger of excess pressure, which might occur with the compressed air apparatus.

As suggested in a preliminary report,[8] amphetamine vapor in the middle ear cavity has proved to be particularly useful in cases of chronic catarrhal otitis media. From 1935 to date more than 300 patients have been so treated. The patients were selected from private practice as well as from the clinics of St. Agnes Hospital, St. Vincent's Hospital and St. Joseph's Hospital and Doctors Hospital, Philadelphia.

Photograph of apparatus used in insufflation of amphetamine vapor into the eustachian tube.

In cases of acute early blocking of the eustachian tube one treatment may suffice to give complete relief. In every case some relief occurs immediately. Conditions of longer duration may require treatments over an indefinite period. Amphetamine vapor promptly constricts the mucosa. The increased pressure in the middle ear cavity tends to overcome the external pressure on the tympanic membrane, with

8. Sulman, L. D.: Certain Conditions in Which a Volatile Vasoconstrictor Has Proved of Particular Value, M. Times & Long Island M. J. **63**:374-377, 1935.

consequent decided improvement in hearing, as registered on the audiogram. A laboratory check showed that about 2 mg. of amphetamine sulfate was given at each treatment, which was obviously insufficient to cause demonstrable systemic reactions. Nor were any secondary local reactions observed. I am convinced that treatment of eustachian tube blockage is shortened by the use of amphetamine vapor.

SUMMARY

Amphetamine vapor applied directly to the eustachian tube is used to relieve eustachian tube blockage. Methods are outlined for introducing amphetamine into the eustachian tube with a catheter or by means of a Politzer bag.

323 South Eighteenth Street.

THE EUSTACHIAN ORIFICE

Relation to Landmarks in the Nose and Nasopharynx

A. MANGIARACINA, M.D., BROOKLYN

Thirty-two adult patients of both sexes were examined to determine the following relations of the eustachian orifice: (1) distance of the orifice from the posterior pharyngeal wall; (2) height of the orifice from the nasal floor; (3) distance of the posterior edge of the septum at the floor of the nose from the posterior pharyngeal wall, and (4) distance from the posterior pharyngeal wall to the floor of the nasal vestibule. In all but 3 cases the examinations were made in Dr. William Herbert's clinic at the Brooklyn Eye and Ear Hospital. The youngest patient was 25 and the oldest 72 years of age.

A nasopharyngoscope was introduced into one side of the nose and held in place with a headband holder. The measurements were carried out through the opposite side. The accompanying table gives the results obtained.

Forty-one measurements were taken to determine the distance of the eustachian orifice from the posterior pharyngeal wall. The longest distance was 20 mm. and the shortest 12 mm., with an average distance of 15.7 mm. The height of the eustachian orifice from the nasal floor was determined thirty-three times. The highest and the lowest values were 12 and 5 mm., respectively, and the average was 8.36 mm.

In the 25 patients examined the shortest anteroposterior diameter of the nasopharynx was 22 mm. and the longest 32 mm. The average distance was 26.5 mm.

For the 17 patients measured the average distance from the posterior pharyngeal wall to the nasal vestibule was 95.3 mm. The longest distance was 100 mm. and the shortest 89 mm.

According to these measurements, the posterior edge of the septum at its base is from 6 mm. (patient A. N.) to 17 mm. (patient J. S.) anterior to the eustachian orifice.

This variation in the relation of the tubal opening to the posterior edge of the septum accounts for the not infrequent failure of the tip of the eustachian catheter to enter the orifice when the posterior edge of the septum is used as a guide.

In the greater number of patients whose cases are summarized in the table the septum was from 10 to 13 mm. anterior to the eustachian opening. The average distance for the patients examined was 11 mm.

In introducing and then rotating a catheter having a curvature of 145 degrees and a beak of 20 mm. and then withdrawing it until it strikes the posterior edge of the septum, the beak of the catheter is found to project about 12 mm. into the nasopharynx. When the catheter is then rotated outward 180 degrees it will be opposite the eustachian opening only in those cases in which the distance of the orifice from the posterior edge of the septum is approximately that of the pro-

Measurements Relative to the Eustachian Orifice in Thirty-Two Patients

Patient	Sex	Age, Years	Distance from Tubal Orifice to Posterior Pharyngeal Wall, Mm.	Height of Orifice from Nasal Floor, Mm.	Distance of Posterior Edge of Septum from Posterior Pharyngeal Wall, Mm.	Distance from Posterior Pharyngeal Wall to Floor of Nasal Vestibule, Mm.
C. W.	M	68	Right 15 Left 15.5	Right 10 Left 11	27	..
R. B.	M	45	Right 17.5 Left 17	Right 8 Left 11	32	..
A. N.	F	36	Right 16	8	22	..
C. O.	F	70	Right 17	9	27	..
M. I.	M	29	Left 17	12	27	..
J. S.	M	54	Right 15.5	6	22	..
A. F.	F	69	Right 14	10	27	..
J. M.	M	58	Right 19	5	25	..
E. E.	M	65	Right 19	12	29	94
W. P.	M	69	Left 18	11	30	100
G. D.	M	50	Left 14	8	27	89
M. N.	M	39	Left 14	8	27	99
F. B.	M	72	Right 16	8	22	92
S. S.	M	65	Right 15	7	29	100
V. V.	M	25	15	9	27	95
M. K.	F	29	13	5	24	95
J. C.	M	65	Right 14	7	22	95
J. B.	M	48	Right 13	8	24	90
G. M.	M	26	Right 17	8	27	97
W. S.	M	63	Left 18	12	27	96
N. D.	M	59	Right 14	10	29	99
J. M.	M	33	Right 15	9	31	97
J. S.	F	33	Right 15	6	32	95
G. C.	M	37	Left 13	8	24	94
G. S.	M	43	Right 12	5	22	94
W. S.	M	40	Right 17.5 Left 18	..	..	..
E. B.	M	26	Right 15 Left 14	..	..	..
M. F.	F	49	Right 16 Left 16	..	..	..
S. M.	M	54	Right 14 Left 14	Right 6 Left 8	..	..
E. B.	M	26	Right 17 Left 15.5	Right 8 Left 8	..	..
P. E.	M	48	Right 16 Left 14	Right 8 Left 7	..	..
R. P.	F	26	Right 20 Left 20	.....	..	..

jecting tip of the catheter, viz., about 10 to 12 mm. As in the greater number of cases the distance is between 10 and 13 mm., one is usually successful in entering the tube when this method is used.

The other common method of catheterization, which utilizes the posterior prominence of the eustachian orifice as a guide, is the better method because here a landmark is used which has a constant relation to the opening. A no. 2 catheter either 12.5 or 15 cm. long is the most serviceable. When one is working with a nasopharyngoscope in the opposite nostril the shorter catheter is more convenient, because it does not get in the way of the eyepiece of the instrument.

Occasionally one is unable to catheterize the eustachian tubes satisfactorily by the usual methods employed. The tubal orifices in such cases can then be treated under direct vision with the nasopharyngoscope inserted in the opposite nostril.

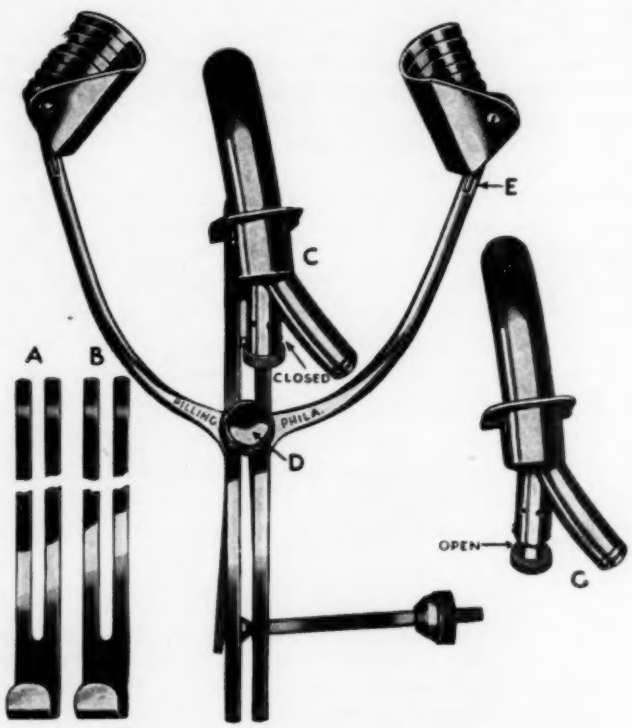
Whenever anatomically possible, the routine use of the nasopharyngoscope is recommended in all examinations of the nose and throat.

685 Bushwick Avenue.

A RETRACTOR FOR USE IN NECK SURGERY

HUGH G. BEATTY, M.D., COLUMBUS, OHIO

A retractor is presented which is adaptable to use in certain operations on the neck, particularly operations on the larynx and especially total laryngectomy. This instrument has finally been perfected to suit my wants after numerous changes



Retractor for use in operations on the neck.

in several previous models. It does away with the presence and irregular aid of another assistant.

It makes possible retraction of the lateral border and the lower angle of the incision and furnishes a nontraumatic means of administering vapor anesthesia, if and when such is desired. It also is quickly adaptable to the insertion of a soft rubber tube for aspiration of the bronchi.

DESCRIPTION OF THE INSTRUMENT

The framework can be easily understood as working on hinge *D* and regulated by the set screw on the handle. *D* is a double deck screw-nut, two thumb screws at different levels. These screws hold in place the anesthetic and aspiration tube, *C*, and the lower angle wound retractor, *A* or *B*. *A* and *B* are lower angle retractors with retracting lips of different depths, one for a lean and the other for a thick neck. For a thick neck, *B* and *C* may be set high; for a thin neck, *A* may be under the lower lug and *C* between the two, about 1 cm. higher or adjusted as desired.

One of the chief advantages of these slotted retractors and the slotted shaft for *C* is that they can be freely bent to suit the thinness or thickness of the particular neck.

The anesthetic tube carries a valve which can be opened or closed when the trachea is tightly closed by the tracheostomy tube for anesthetic purposes. This unit is easily and quickly detached from the tracheostomy tube at a connection for aspiration, leaving an open tube firmly held in the larynx or tracheal stump.

The respiration, anesthetic and aspiration cannula can be used either in the exposed larynx, when the larynx is detached first from the esophagus, or in the tracheal stump, when the removal is carried from below up. This is made possible because the shafts *A*, *B* and *C*, which fit under set screws at *D*, are flexible and can be bent up or down and shortened or lengthened (by adjustment) to fit the individual situation.

The joint at *E* does away with the rigid retraction which always is a nuisance in the ordinary neck retractors.

Bending of the shafts of *A*, *B* and *C* does not injure the instrument.

The handles of the instrument lie flat on the chest and are well out of the way of the operation.

150 East Broad Street.

Progress in Otolaryngology

Summaries of the American and British Bibliographic Material Available in the Field of Otolaryngology

TONSILS AND ADENOIDS

J. DUDLEY SINGLETON, M.D.
DALLAS, TEXAS

In reviewing the American and English literature for 1940 and through September 1941 on the subject of tonsils and adenoids one finds several interesting and worth while articles. However, there has been little research or few results of systematic studies reported in this important field of otolaryngology. There has been considerable interest in postoperative complications and infections involving the tonsils. A few articles on the employment of anesthesia in tonsillectomy and several papers dealing with tumors of the tonsils have appeared.

TONSILS

General Considerations.—Reimann and Havens [1] state that the concept of focal infection in relation to systemic disease is firmly established, but that the causative relationship of infections about the teeth and tonsils to systemic disease is unproved and that the removal of teeth and tonsils in an effort to influence the course of systemic disease is unjustified in the majority of cases. They cite the criteria for the diagnosis of chronically diseased tonsils and point out that local evidence of disease often disappears if recovery from the general disease occurs and that tonsils, particularly in malnourished, underweight or otherwise subnormal children, probably would become "normal" in appearance spontaneously if the general health were improved by hygienic measures. They conclude their article as follows:

> (*a*) The theory of focal infection, in the sense of the term used here, has not been proved, (*b*) the infectious agents involved are unknown, (*c*) large groups of persons whose tonsils are present are no worse than those whose tonsils are out, (*d*) patients whose teeth or tonsils are removed often continue to suffer from the original disease for which they were removed, (*e*) beneficial effects can seldom be ascribed to surgical procedures alone, (*f*) beneficial effects which occasionally occur after surgical measures are often outweighed by harmful effects or no effect at all, and (*g*) many suggestive foci of infection heal after recovery from systemic disease, or when the general health is improved with hygienic and dietary measures.

1. Reimann, H. A., and Havens, W. P.: Focal Infection and Systemic Disease: A Critical Appraisal, J. A. M. A. **114**:1-6 (Jan. 6) 1940.

Renaud,[2] in a series of articles, states that with functioning tonsils middle ear infection, mastoid involvement and sinus disease would be rare indeed. He then proceeds to describe an unorthodox method of treating tonsils, as follows:

> In the first years of using [my] method, I depended mainly upon pressure firmly applied to the anterior pillar well back of the tonsils. The débris, bacteria and toxins removed from the tonsils by this effort will be followed by mucous secretions from nasal passages and sinuses, upon which the great benefits from tonsil treatment depend. . . . In occasional refractory cases, however, it was found that further benefit could be secured by slight slitting of the areas especially affected, followed by exudation of a few drops of blood. Later, the advantages of using a simple syphoning of the nasal passages at this stage became apparent, due to the fact that the ostia are now freely open. . . . A thorough cleansing of all the nasal cells and sinuses is thus secured. Tincture of iodine thirty-three and one-third per cent in glycerine is used as an application to the tonsils after the treatment described above. For secretions of a more purulent character, the application of silver nitrate, 20 to 180 grains to an ounce, applied carefully to areas affected, is used.
>
> Occasional obstinate and severe cases will be further benefited by infraction of the middle turbinate. I almost make this procedure a routine in my hay fever and asthma work in adults. . . . Septal deviation should be left alone unless very marked. Naturally, polypoid tissue requires attention.
>
> I see no reason why the simpler operation of tonsillotomy could not be used for reduction in size of large tonsils which do not respond, in this respect, to treatment.

With his treatment the author claims satisfactory results in such conditions as bursitis, Hodgkin's disease, pellagra, encephalitic lethargica, leukoplakia, sacroiliac synchondrosis, leukemia, asthma, tic douloureux, diabetes, insomnia, nervous disorders, Bell's palsy, gastrointestinal troubles, otitis media, vertigo and iritis.

Paul and Galloway,[3] on the basis of the study of 843 pregnancies occurring in 704 women, are convinced that foci of infection in teeth and tonsils should be carefully searched out and removed as soon as found. Failure to remove these foci of infection seems to increase the incidence of abortion and perhaps is a factor tending to increase pyelitis. The incidence of abortion resulting from extraction of teeth or removal of the tonsils is no greater than that in apparently normal women, and fear of removing them should not be a deterring force. This study shows that 4.27 per cent of the series had tonsils which could be considered as harboring foci of infection. The incidence of abortion which could be attributed to the removal of foci was 3.5 per cent for extraction of infected teeth and 4.3 per cent for removal of tonsils. These figures are lower than the incidence for the entire group.

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2. Renaud, G. L.: Tonsils, M. Rec. **152**:239-242 (Oct. 2); 279-282 (Oct. 16); 317-338 (Nov. 6) 1940; **153**:155-157 (March 5) 1941.

3. Paul, T. D., and Galloway, C. E.: Focal Infection in Pregnancy, Am. J. Obst. & Gynec. **39**:694-697 (April) 1940.

Barrington-Ward [4] discusses at length the indications for removal of the tonsils and adenoids. He considers the tonsils a more dangerous factor because their position and structure allow organisms to lie dormant or to remain moderately but continuously active, leading to chronic general poisoning or to flare-up of acute inflammation at indefinite intervals. The adenoids act mechanically by obstructing normal respiration or by harboring a low grade infection, causing inflammation of the ear or of certain cervical glands. If only the adenoids are removed and septic tonsils are left behind, the adenoids are likely to grow again. The writer calls attention to the fact that the presence of septic tonsils is not always easy to determine and states that the secondary effects should be taken into consideration in making a diagnosis.

Shea [5] briefly reviews the component parts of Waldeyer's ring and calls attention to the ages during which the various parts are most active. The signs and symptoms of adenoids are discussed briefly. Errors in surgical removal of tonsils and adenoids are discussed in detail under several headings, as follows: Thornwaldt's disease; anesthesia; incision of abscess; operation; hemorrhage; accidents during operation; post-operative care; infection; deep cervical infection; symptoms of deep cervical infection; post-tonsillectomy pulmonary abscess; prevention of pulmonary abscess; adenoids; bad results.

In conclusion, Shea states that the law of operative procedures on the tonsils and adenoids should be the complete removal of the tissue—"all this tissue and nothing but this tissue." This paper is well worth reading.

Kaiser [6] states that the problem that confronts the physician today is: "When should the tonsils be considered a menace to a child's development?" In a study of 4,400 children over a period of ten years the incidences of common infection of the upper respiratory tract, bronchitis, pneumonia and the various manifestations of rheumatic infection were recorded. Tonsillectomy was advised for all of the 4,400 children, but only half of the group submitted to the operation. None of the children in the control group of 2,200 were operated on during the succeeding years. As a result of his studies, Kaiser found that markedly hypertrophied tonsils and tonsils that are repeatedly inflamed, giving rise to attacks of tonsillitis and cervical adenitis, frequently impair normal physical development and that such infections as the common cold, otitis media, sinusitis and laryngitis may unfavorably influence the

4. Barrington-Ward, L.: Indications for Removal of Tonsils and Adenoids in Children, Proc. Roy. Soc. Med. **33**:347-356 (April) 1940.

5. Shea, J. J.: Frequent Errors in Tonsil and Adenoid Surgery, Mississippi Doctor **18**:143-151 (Aug.) 1940.

6. Kaiser, A. D.: Significance of the Tonsils in the Development of the Child, J. A. M. A. **115**:1151-1156 (Oct.) 1940.

child's normal development. But he was unable to demonstrate that the tonsils are often a causative factor in these infections. Removal of the tonsils does not appear to reduce the incidence of bronchitis, pneumonia or tuberculosis. By reducing the number of infections of the throat tonsillectomy appears to benefit the rheumatic child. Kaiser concluded his article by stating that in approximately 20 per cent of children the tonsils are either hypertrophied or diseased sufficiently to have an unfavorable influence on the development of the child and that such tonsils should be removed.

Whedon,[7] in an article based on the records of 1,500 tonsillectomies over the period from 1906 to 1940, states that complete removal of all diseased tonsils is essential to good health. In his experience he has found that children 6 years of age can be successfully operated on with local anesthesia for the removal of tonsils and adenoids. I should be hesitant in undertaking such an operation on any child under 12 or 14 years of age.

Hansel and Chang,[8] in a study of the relation of allergy and tonsillectomy in children, summarize their article as follows:

1. In the management of frequent colds, sinusitis, bronchitis and recurrent pneumonia the possibility of allergy as an etiologic factor must always be considered.

2. Of 200 children considered as a routine for removal of the tonsils and adenoids because of these complaints, a definite diagnosis of nasal allergy was established in twenty six, or 13 per cent. It is significant that 6 of these patients had hay fever.

3. These cases of respiratory allergy may be overlooked unless an adequate history, physical examination and cytologic studies of the nasal secretion are carried out in each instance.

4. Removal of the tonsils and adenoids in allergic children should not be performed during hay fever seasons, and the indications for operation should be the same as in nonallergic children.

5. The tonsils and adenoids should not be removed with the idea of alleviating allergic symptoms.

Layton[9] states that when a young person completely changes his mode and site of living the hygiene of the upper respiratory tract is altered and will result in a series of sore throats resembling tonsillitis. He calls this condition "evacué's sore throat" and advises that such persons should not have the tonsils removed until they have been in the new area for two years. "Shelter tonsillitis" he describes as occurring in the late fall and early winter as a result of crowding together in poorly

7. Whedon, E.: Tonsillectomy Experiences in Private Practice, Rocky Mountain M. J. **38**:29-33 (Jan.) 1941.

8. Hansel, F. K., and Chang, C. S.: Relation of Allergy and Tonsillectomy in Children, Arch. Otolaryng. **31**:45-52 (Jan.) 1940.

9. Layton, T. B.: Evacuee's Sore Throat and Shelter Tonsillitis, Brit. M. J. **2**:531 (Oct. 19) 1940.

ventilated rooms. The lymphoid tissues swell and become red, the reaction being often pronounced in the tonsils.

Viola [10] presents a case in which the patient had a history of bleeding from one tonsil as well as hemorrhages over the body after light trauma. After exclusion of the usual causes of hemorrhage and in view of a gynecologic report of infantile uterus and maldevelopment of the ovaries, the patient was given estrogen for three months with satisfactory results.

Thomas and Goldstein [11] state that there is nothing characteristic about the sore throat of secondary syphilis. It may present any picture, from mere enlargement of the tonsils or mild inflammation to deep ulceration or a thick membranous exudate. They urge that every patient with chronic tonsillitis be given a Wassermann test and a thorough examination for stigmas of secondary syphilis.

Rosencrantz and Hurwitz,[12] in a study of 103 female tuberculous children, found the incidence of tuberculosis of the tonsils to be approximately 18 per cent. The tuberculous process in the tonsils was latent; there was no macroscopic change in the tonsils that made a clinical diagnosis of the condition possible. Of the 19 patients with tonsillar tuberculosis, 18 had tubercle bacilli in the gastric contents, as revealed by guinea pig inoculation. No theory as to the exact mechanism of tonsillar infection in these patients was offered. Indications for tonsillectomy were the same as in the nontuberculous patients. The authors believe that removal of the tonsils in these children will reduce absorption from tuberculous foci and perhaps offer a prophylactic measure against the development of tuberculous cervical adenitis.

Peluse [13] has devised an ether tube and lip retractor attachments for standard type mouth gags and finds them to be very useful in performing tonsillectomies with general anesthesia. The attachments are described and illustrated.

Matis [14] describes and illustrates the use of a new instrument which he has devised for suturing in the presence of profuse hemorrhage following tonsillectomy. He finds the instrument to be effective in controlling severe hemorrhage.

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10. Viola, N.: Bleeding from the Tonsils Caused by Lack of Hormone, J. Laryng. & Otol. **55**:382-384 (Aug.) 1940.

11. Thomas, E. W., and Goldstein, D. H.: Chronic Tonsillitis in Secondary Syphilis, New York State J. Med. **41**:256-259 (Feb. 1) 1941.

12. Rosencrantz, E., and Hurwitz, S.: Tuberculosis of Tonsils, Am. Rev. Tuberc. **43**:388-393 (March) 1941.

13. Peluse, S.: Ether Tube and Lip Retractor Attachments for Mouth Gag, Arch. Otolaryng. **32**:121 (July) 1940.

14. Matis, E. I.: A New Method of Suturing in the Treatment of Profuse Hemorrhage Following Tonsillectomy, Arch. Otolaryng. **33**:86-88 (Jan.) 1941.

Vistreich [15] describes and illustrates the use of a grasping forceps of the alligator jaw type in securing the bleeding point during tonsillectomy. A small rubber disk which has a puncture hole in its center is then slipped over the slender grasping end of the forceps to encircle the tissue held. The author claims that if properly applied the elastic tie will remain in position several days. It may readily be removed with a grasping forceps at the end of forty-eight hours.

Johnson and Palmer [16] treated 20 unselected patients by roentgen irradiation of the tonsils to determine the effect of roentgen rays in eradicating or reducing the tonsillar tissue. The patients were followed at stated intervals for one year after treatment. It was the authors' conclusion that roentgen treatment in the dose given was not effective in reducing or eradicating tonsillar tissue.

Bacteriology.—MacDonald, Simmons and Keogh [17] point out that the serologic classification of hemolytic streptococci introduced by Lancefield enables a distinction to be made between strains which as pathogens or potential pathogens of man are of epidemic importance and those which are harmless saprophytes or opportunist pathogens, and that the pathogenic group A strain is found primarily in the human throat, especially in the tonsils. They state that the source of the streptococci found in puerperal and surgical streptococcic infections is therefore probably the human tonsil.

Chesterman and Scandrett [18] report the results of studies on the incidence of hemolytic streptococci in the throats of resident nurses in the Women's Hospital of Sydney, Australia, the effect of the presence or absence of tonsils and the methods of eradicating the infection. Cultures of material from the throats of 297 nurses with tonsils or tonsil tags showed 7 per cent to be carriers at the time their first cultures were made, whereas of 145 nurses without tonsils only 1.4 per cent were carriers in the beginning. Of the nurses whose tonsils were present or were incompletely removed 22 per cent were at some time carriers, but of the nurses whose tonsils were completely absent only 9.7 per cent were affected. Tonsillectomy was successful in eliminating the infection in 26 of 29 patients, and administration of sulfanilamide in adequate

15. Vistreich, F.: Easy Ligation of Blood Vessels in Connection with Tonsillectomy, Arch. Otolaryng. **31**:684-686 (April) 1940.

16. Johnson, C. I., and Palmer, R. S.: The Effect of X-Radiation on Tonsillar Tissue, Ann. Otol., Rhin. & Laryng. **49**:755-758 (Sept.) 1940.

17. MacDonald, I.; Simmons, R. T., and Keogh, E. V.: Further Observations on the Incidence of Haemolytic Streptococci in the Tonsils, M. J. Australia **1**:849-851 (June 22) 1940.

18. Chesterman, J. N., and Scandrett, S.: The Incidence of Haemolytic Streptococci in the Throats of Obstetric Nurses: Results of Treatment by Tonsillectomy and Sulphanilamide, M. J. Australia **2**:695-699 (Dec. 28) 1940.

doses under careful supervision was found to be a valuable method of treatment when the other measures had failed.

Anesthesia.—Lillie, Pastore and Mansel [19] point out that the choice of anesthesia for tonsillectomy and intranasal operations should not be left entirely to the patient. Contraindications to operation and to various anesthetics are discussed briefly. The authors state that some physicians do not use local anesthesia successfully because they are unfitted to do so and that there are some physicians who do not realize, or will not recognize, their limitations and as a result condemn the method as used by others. They conclude the paragraph by asking the question: "What could be more tragic than an irritable, incompetent surgeon operating on an excited, resisting, noncooperating patient?" They discuss briefly the types of patients encountered and the preoperative care of the patient and describe in detail the technic of administering a local anesthetic for tonsillectomy.

Meyersburg [20] discusses the preliminary preparation of a patient for local anesthesia in tonsillectomy and describes in detail his technic of injection of the local anesthetic as follows:

> The mucous membrane of the anterior pillar is superficially infiltrated. This may be accomplished with a single puncture. The needle is next engaged in the mucosal supratonsillar reflection on the same side and, as injection proceeds from above downward, the mucous membrane of the posterior pillar is infiltrated until the structure stands out in relief like an actual column. The posterior pillar on the opposite side is treated similarly. The first tonsil to be removed is grasped with a tonsil forceps nearer the upper pole and displaced medially until a sulcus appears laterally. This dimple corresponds pretty much to the space between the layers of pharyngeal fascia. It is here that the first deep injection is made about two-thirds of the distance from the top of the tonsil since most of the nerve and blood supply enters the tonsil at this point. The final deep injection is made low down in the same vertical plane, anesthetizing the trunk and branch and facilitating bloodless and painless dissection down to the lateral tongue base border. The technic is repeated for the operation on the second tonsil after the first has been removed.

Cagan and Macedonia [21] made a clinical study of the value of local application of neo-synephrin hydrochloride, 1 per cent, in tonsillectomy and demonstrated its ability to reduce markedly the amount of mucus. Since this drug is only slightly less effective than atropine, does not have to be given hypodermically and exerts only a local effect, in contrast to atropine, 1 per cent neo-synephrine hydrochloride in the form

19. Lillie, H. I.; Pastore, P. N., and Mansel, L. H.: Methods for Producing Local Anesthesia for Tonsillectomy, Intranasal Operations and Operations on the Paranasal Sinuses, Ann. Otol., Rhin. & Laryng. **49**:38-51 (March) 1940.

20. Meyersburg, H.: Technic of Local Anesthesia in Expanded Tonsillectomy, M. Rec. **151**:231-232 (April 3) 1940.

21. Cagan, A., and Macedonia, D.: The Use of Neosynephrin Hydrochloride in Tonsillectomies, M. Rec. **151**:170-171 (March 6) 1940.

of a spray may be safely used in tonsillectomy. The authors are now using it routinely in all operative cases in which the open drop method of inducing ether anesthesia is employed.

Zisserman and Cohen [22] report using paraldehyde as a preoperative sedative for tonsillectomy in children in 224 cases. In administering the drug, the patient is given a simple enema on the morning of the operation. One hour preceding the operation a retention enema of paraldehyde is administered. According to the authors, best results are obtained with 1 cc. of paraldehyde for each 5 pounds (2.3 Kg.) of body weight, mixed with an equal volume of tap water and given with a soft rubber catheter and syringe at room temperature. They have observed no bad results from the use of the drug.

Complications.—Matis [23] calls attention to the importance of post-tonsillitis complications and points out that postanginal sepsis is the most dangerous. He states that it carries a death rate of 5.3 per thousand. He reports 2 fatal cases with septic thrombophlebitis and multiple metastatic abscesses.

Fleet [24] reports 3 cases of jugular thrombosis following tonsillitis. In his article he gives a brief summary of the literature on this condition and discusses the treatment of the infection in some detail. If improvement does not occur after opening the internal jugular vein, Fleet advises drainage of the lateral sinus.

Houser and Fitz-Hugh [25] report a case of pulmonary abscess following tonsillectomy in which abortion was induced with sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine). The diagnosis was made on the basis of the physical, laboratory and roentgenographic findings. The patient's condition had not improved with sulfanilamide therapy. After a blood transfusion and the institution of sulfapyridine therapy the patient showed definite improvement within twenty-four hours, and within ten days the symptoms subsided and the clouding in the lung disappeared. The authors believe that sulfapyridine may be of great value in the treatment of postoperative pulmonary suppuration if employed in the "pneumonic stage," before gross cavity formation occurs.

Scherf [26] is of the opinion that during and immediately after acute tonsillitis damage of the myocardium occurs more frequently than is

22. Zisserman, L., and Cohen, H. B.: Paraldehyde: A Basal Anesthetic for Tonsillectomy in Children, Am. J. Surg. **50**:506-508 (Dec.) 1940.

23. Matis, E. I.: Two Unusual Cases of Postanginal Sepsis, Ann. Otol., Rhin. & Laryng. **49**:559-563 (June) 1940.

24. Fleet, R. L.: Jugular Thrombosis After Tonsillitis, Brit. M. J. **2**:223 (Aug. 16) 1941.

25. Houser, K. M., and Fitz-Hugh, T., Jr.: Post-Tonsillectomy Pulmonary Abscess (Preabscess Stage) Aborted with Sulfapyridine, Arch. Otolaryng. **31**: 855-857 (May) 1940.

26. Scherf, D.: Myocarditis Following Acute Tonsillitis, New York M. Coll. & Flower Hosp. Bull. **3**:252-260 (Dec.) 1940.

usually suspected and states that the complication deserves more attention than it often receives. Some statistics show that as high as 67 per cent of patients with acute tonsillitis show evidence of myocardial involvement. On the basis of his experience the author believes that 10 to 15 per cent of patients who suffer from acute tonsillitis manifest symptoms, signs or electrocardiographic changes which suggest involvement of the myocardium. He states that the prognosis is good, that treatment consists of rest and that tonsillectomy should not be performed until at least six weeks after the signs of activity have subsided.

Hadley [27] reports a case of scarlet fever occurring in a child on the third day following a tonsillectomy. Investigation revealed that scarlet fever was prevalent in the school the child was attending, and the author concluded that he was dealing with a case of ordinary scarlet fever coinciding with convalescence from tonsillectomy.

Youngerman [28] calls attention to the high mortality rate of diphtheria in adults associated with peritonsillar edema and briefly discusses the differential diagnosis between it and a peritonsillar abscess. He points out that surgical intervention greatly increases the mortality rate and strongly advises against incising a peritonsillar swelling without first making a thorough differential diagnosis.

In discussing peritonsillar abscesses Brighton [29] advises tonsillectomy with the use of general anesthesia in those cases in which the abscess is deep seated and is not reached by simple incision at the upper pole of the tonsil. He reviews the practical anatomy of the fascial spaces of the neck and discusses the diagnosis and treatment of abscesses that have extended into these spaces.

Krill and Toomey [30] report the occurrence of 5 cases of the bulbar type of poliomyelitis in a family of 6 children following general tonsillectomy. Symptoms of the disease developed twelve days after operation. The youngest child, aged 2½ years, was not operated on and did not contract the disease. Three of the children died.

Fischer, Stillerman and Marks [31] state that in the Toronto epidemic of 1937 acute poliomyelitis occurred more often among children between the ages of 3 and 12 years who had recently been tonsillectomized than

27. Hadley, H. G.: Scarlet Fever Following Tonsillectomy, Laryngoscope **51**:269-271 (March) 1941.

28. Youngerman, W. N.: Peritonsillar Edema in Diphtheria: Danger of Incision, Illinois M. J. **78**:169-171 (Aug.) 1940.

29. Brighton, G. R.: Peritonsillar Abscess: Complications and Treatment, S. Clin. North America **20**:557-565 (April) 1940.

30. Krill, C. E., and Toomey, J. A.: Multiple Cases of Tonsillectomy and Poliomyelitis, J. A. M. A. **117**:1013 (Sept. 20) 1941.

31. Fischer, A. E.; Stillerman, M., and Marks, H. H.: Relation of Tonsillectomy and of Adenoidectomy to the Incidence of Poliomyelitis, Am. J. Dis. Child. **61**:305-321 (Feb.) 1941.

among those who had not been operated on. They also call attention to the fact that among the children with poliomyelitis the incidence of the bulbar type was more than twice as great in those whose tonsils had been removed at any time previously as in those whose tonsils had not been removed.

Helms and Willcocks [32] call attention to the high incidence of the bulbar type of poliomeylitis following tonsillectomy and adenoidectomy and state that there is strong evidence in support of the contention that operations on the nose and throat favor the development of poliomyelitis of the bulbar type. The authors caution against such operations when the disease is prevalent.

Longworthy [33] believes that the effort being made to connect the avenue of infection in poliomyelitis with trauma following removal of the adenoids and tonsils is unwise. He supports his opinion by citing other authors who suspect poliomyelitis to be an "intestinal disease" since the virus can be isolated readily from patients' stools and only with great difficulty from the nose.

Tumors, Benign and Malignant.—Davis [34] discusses briefly the classification and diagnosis of sarcoma of the tonsil and calls attention to the fact that the condition is rare. He describes three clinical types. The first and most common is characterized by sudden, rapid enlargement of one tonsil, with equally rapid enlargement of the corresponding lymphatic glands of the neck in a patient about 40 years of age. The second type resembles severe Vincent's angina when first seen. The ulceration, at first superficial and confined to the tonsil area, later becomes very deep and extends to the surrounding tissues. Glandular enlargement in the neck is slight and not a distinctive feature. The third type is characterized by early enlargement of the cervical glands. In the early stage of this type the primary growth in the tonsil may not be apparent but later becomes manifest. Davis points out that lymphosarcoma is very radiosensitive and stresses the importance of early diagnosis and radium treatment.

Mattick [35] reports a series of cases of cancer of the tonsil covering the period from 1915 to 1935. His records show that during these

32. Helms, K., and Willcocks, W. I.: The 1937-1938 Epidemic of Acute Anterior Poliomyelitis in New South Wales with Special Reference to the Change in Age Incidence: Poliomyelitis During Pregnancy and Poliomyelitis Following Tonsillectomy, M. J. Australia **1**:467-476 (April 19) 1941.

33. Longworthy, H.: Are Unwise Efforts Being Made to Connect the Avenue of Infection in Poliomyelitis to Trauma Following Adenoid and Tonsil Operation? Laryngoscope **50**:1100-1104 (Nov.) 1940.

34. Davis, E. D. D.: The Clinical Aspect of Lymphosarcoma of the Tonsil, Proc. Roy. Soc. Med. **34**:679-683 (Aug.) 1941.

35. Mattick, W. L.: The Diagnosis and Treatment of Cancer of the Tonsil, Radiology **35**:268-273 (Sept.) 1940.

years cancer of the tonsil had an incidence of approximately 1 per cent of all cancer and constituted 10 per cent of all intraoral cancer. Of the cases studied, 91 per cent were in males, and the period of greatest frequency was in the fifth and sixth decades, in which approximately two thirds of the cases were observed. In making a diagnosis, Mattick thinks that simple inspection of the palatine fauces will generally disclose the condition but visual examination should always be accompanied by palpation with the gloved finger, since induration commonly associated with cancer is probably one of the earliest presumptive evidences of the condition. The finding of metastatic nodes in the neck should always place on the examiner the responsibility of making an exhaustive search of the upper respiratory passages for a primary source. Biopsy of either the primary lesion or the secondarily involved glands should be resorted to for confirmatory pathologic evidence in all cases and does no harm provided adequate treatment follows soon after. According to the author, the most common tumor of the tonsil is squamous cell epithelioma, with or without pearl formation. Next are the transitional forms of lymphoepithelioma of the Schmincke or Regaud type, and least common are adenocarcinoma and the spindle cell anaplastic types. He states that the therapeutic management is the concern of a competent radiologist.

Martin and Sugarbaker[36] report on 157 unselected, consecutive cases of cancer of the tonsil, including those of all patients with all stages of the disease who applied to the Memorial Hospital in New York city during the years 1931 to 1935 inclusive. The authors use the term "cancer of the tonsil" to designate all "those growths which arise in the palatine tonsil itself, at its periphery in the tonsillar fossa, in the tonsillar pillars, or in the plica triangularis." The authors discuss in detail the etiology, symptoms and clinical course, diagnosis, method of treatment, end results and prognosis of the disease. They report a five year cure rate of 18 per cent. The article includes a bibliography of fifty-three references.

Dunn,[37] in a paper on benign and malignant tumors of the tonsil and peritonsillar area, points out that papilloma is the most frequent of all benign tumors in this area. He states that malignant disease in this region is rather uncommon but that it is nearly always primary when it does occur. The tumor may be sarcoma, lymphosarcoma, epithelioma, scirrhous carcinoma or adenosarcoma. The author states that the prognosis of all malignant growths in the tonsil or the peritonsillar region is exceedingly poor when treated surgically but that it is somewhat more encouraging when irradiation is used; he states further that the

36. Martin, H., and Sugarbaker, E. L.: Cancer of the Tonsil, Am. J. Surg. **52**:158-196 (April) 1941.

37. Dunn, L. S.: Tumors, Benign and Malignant of the Tonsil and Peritonsillar Area, Hebrew M. J. **2**:179-183, 1940.

outlook in cases of sarcoma, when the tumor is diagnosed and treated early, is much more hopeful than in cases of carcinoma.

Young and Price [38] present a case of a diabetic woman aged 69 who complained of a choking sensation and a lump on the right side of the neck. Examination revealed the right tonsil to be enlarged, and there was an enlarged gland, of firm consistency, behind the angle of the mandible on the right side. On the left side there was a similar, but smaller, swelling of the tonsil. The tonsils were excised with the use of chloroform anesthesia, and the patient made an uninterrupted recovery. Her general condition has remained satisfactory to date, two and a half years after treatment. The pathologic diagnosis of both tonsils was lymphosarcoma.

Cosco and Pohlmann [39] report a case of primary lymphosarcoma of the tonsil and stress the importance of biopsy of all suspicious lesions involving the tonsils. The diagnostic work and treatment with radium implants were carried out in the office, with satisfactory results.

O'Donnell [40] reports a case of a girl 6 years of age. Attached to the lower pole of the left tonsil was a pedunculated tumor. It was of firm consistency and was covered with apparently normal buccal mucosa. The growth was removed, and the report of the microscopic examination was as follows:

> The "tumour" is covered with squamous epithelium, beneath which is a group of round cells, and then a layer of fibrous tissue. It consists of groups of salivary gland alveoli arranged around the periphery, with the center of the mass consisting of adipose tissue, through which run small strands of muscle. The pedicle connecting the mass to the tonsil is composed of muscle tissue.

No diagnosis was made.

ADENOIDS

Negus [41] discusses the defensive mechanism of the nose and nasopharynx and reviews the anatomy of the nasopharyngeal musculature and the anatomic relations of the adenoid tissue. He states that the efficient removal of the adenoids is a more difficult procedure than removal of the tonsils. In order to produce a minimum of trauma in the removal of the adenoids, Negus passes a gloved finger back of the soft palate and slips it up between the eustachian orifice and the adenoid mass, pressing the latter toward the midline of the nasopharynx, and the same maneuver is repeated on the opposite side. He then uses a

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38. Young, S., and Price, L. W.: Bilateral Lymphosarcoma of the Tonsils, J. Laryng. & Otol. **55**:463 (Oct.) 1940.

39. Cosco, N. P., and Pohlmann, H. T.: Primary Lymphosarcoma of the Tonsil, New York State J. Med. **41**:613 (March 15) 1941.

40. O'Donnell, J. H.: A Case of a Simple Tumour of Unusual Nature Attached to the Tonsil, J. Laryng. & Otol. **55**:254 (May) 1940.

41. Negus, V. E.: Adenoids, with Reference to Methods of Removal, J. Laryng. & Otol. **56**:119-129 (April) 1941.

La Force type of adenotome for removal of the adenoid tissue, going back with the instrument if necessary for the removal of portions that may have been left. It is his opinion that curets should not be used in the nasopharynx because they produce too much trauma.

Adams [42] describes four main types of pathologic adenoid tissue which he thinks have an influence on sinal infection in children. In two of the types there is rather pronounced hypertrophy of the adenoid tissue, with blocking of the posterior choanae and interference with normal ventilation and drainage of the sinuses. Adams thinks that these two types have a direct local effect on sinal infection. In the other two types the hypertrophy is characteristic but much less extensive. There is no interference with breathing. He thinks these types harbor chronic infection and have an indirect effect on sinal infection by reducing the patient's general resistance. The author describes the various types of adenoids in some detail and advises their surgical removal.

Calthrop [43] advocates the use of the roentgenogram in detecting adenoid masses that may have been left in operations for their removal. He states that when other methods present insurmountable difficulties, a roentgenogram of the nasopharynx may show an opacity due to soft tissue in the region of the adenoids. According to the author, examination with the use of an anesthetic confirmed this, and in each instance a mass of adenoid tissue was surgically removed and the symptoms were relieved. He does not claim that it is possible to show small pieces of tissue left behind by incomplete removal of the adenoids, but a large or moderate mass of adenoid tissue should be easily visible in the nasopharynx with this method.

Guggenheim [44] describes his technic in performing a direct adenoidectomy and illustrates several instruments that he uses in performing the operation.

Burnam [45] discusses the relative merits of the roentgen ray and radium or radon in reducing or removing adenoid and hyperplastic lymphoid tissue about the pharyngeal orifice of the eustachian tube for relief of or improvement in impaired hearing in children. He favors the use of radon and describes a radon-containing applicator which he employs in his work. The technic and dosage of the treatments are described and discussed in detail.

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42. Adams, W. S.: The Influence of Adenoids on Nasal Sinusitis in Children, J. Laryng. & Otol. **56**:133-146 (April) 1941.

43. Calthrop, G. T.: Radiological Demonstration of Adenoids, Lancet **1**:1005 (June 1) 1940.

44. Guggenheim, L.: Direct Adenoidectomy, Arch. Otolaryng. **33**:314-315 (Feb.) 1941.

45. Burnam, C. F.: Irradiation Treatment of Hyperplastic Lymphoid Tissue, Laryngoscope **50**:663-670 (July) 1940.

Abstracts from Current Literature

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AN ANATOMICAL STUDY OF THE NASOPHARYNX AS A PATHWAY TO THE INFRA-CAROTID PORTION OF THE PETROUS PYRAMID. B. RUSSELL BURKE, Ann. Otol., Rhin. & Laryng. **49**:924 (Dec.) 1940.

Petrositis is discussed in its various aspects, some of the literature is reviewed and cases are cited. The different approaches to the petrosa are discussed. The diagnosis may be difficult. Headache, radiating mostly to the temporal region and anteriorly to the eye on the diseased side, is the most constant and characteristic symptom. The pain is intense and neuralgic. Fever is an inconstant feature and is usually of a low grade septic type. Normal temperature for two or three successive days is not unusual in cases of the less severe form. Paralysis of the sixth nerve does not always occur, but if it does, in association with other symptoms, it should be considered as indicative of the infection involving the tip, or infracarotid portion, of the pyramid. This paralysis may be brought on by swelling and congestion of the dura over Dorello's canal. Roentgenograms should always be made. The author mentions the Vernet, or jugular foramen, syndrome. This is characterized by difficulty in swallowing, anesthesia or hyperesthesia of the soft palate and pharynx, loss of taste in the posterior part of the tongue (glossopharyngeal nerve), paralysis of the vocal cord on the affected side (vagus nerve) or disturbance of function of the sternocleidomastoid and trapezius muscles (sinal accessory nerve). An inflammatory process in the antelabryinth may involve one or all three of these nerves. The author feels that this syndrome is one of the most important diagnostic signs brought to one's attention.

If a diagnosis of petrositis has been made and the mastoid wound revised, with a search for fistulas, with no result, the nasopharyngeal approach should be employed. Nasopharyngeal examinations should be made at regular intervals, particularly if symptoms indicate the presence of anterior petrositis. Examination should be made with a nasopharyngoscope and by palpation with a gloved finger to determine the presence and degree of swelling over the attachment of the longus capitus muscle and the degree of pain or tenderness in this region.

The objective in the surgical procedure described is the plate of bone forming the floor of the carotid canal. General anesthesia is preferred. An incision is made through the soft palate to the right or left of the uvula, according to the side of involvement, and is carried vertically all the way through the soft palate. The whole nasopharynx is exposed when the palate is then retracted. The medial pterygoid plate is palpated and followed downward to the basiocciput. On moving the finger slightly forward the petrobasilar fissure can be felt. Just laterally the inferior aspect of the petrosa is easily located. A vertical incision of 2 cm. is made here, which reaches to the bone. The tissues are retracted, and the bone is exposed for 1 to 2 cm. between the carotid canal and the lacerated foramen. If disease is present, pus or softened bone is found and adequate drainage secured.

This is a preliminary anatomic report, and no clinical results are given.

M. V. MILLER, Philadelphia.

THE TREATMENT AND PREVENTION OF DEAFNESS IN CHILDREN. J. E. BORDLEY, South. M. J. **33**:1159 (Nov.) 1940.

The author believes that children of early school age should have a careful examination of the hearing, as well as a general physical examination, if preventable impairment of hearing is to be controlled. Otoscopic, nasopharyngoscopic and audiometric examinations should be carried out. The perception of high tones, especially, should be tested.

The immediate eradication of infected lymphoid tissue should be undertaken whenever it is found to involve the eustachian tubes. Allergic manifestations should be corrected. Simple tonsillectomy and adenoidectomy are often not enough and should not be performed unless there are definite indications, because in many cases they do not remove the cause of the difficulty in hearing.

These measures, combined with careful treatment of acute infections of the middle ear and the mastoid with such drugs as sulfanilamide and sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) and, when necessary, conservative surgical procedures, offer untold possibilities for the prevention of deafness in later life. All children with any signs of impairment of hearing should have periodic check-up examinations.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

THE SURGICAL TREATMENT OF MÉNIÈRE'S DISEASE. W. E. DANDY, Surg., Gynec. & Obst. **72**:421 (Feb. 15) 1941.

Two essential features of Ménière's syndrome are dizzy spells and deafness. Other symptoms such as nausea, vomiting and tinnitus, may be present. The dizziness is of a definite character—objects rotate or jump rapidly. The attacks are recurrent; they vary in severity and may last for seconds, hours or days. The deafness usually appears slowly and insidiously, attacking all tones more or less but progressing to levels at which for practical purposes hearing is lost.

Dandy believes that the lesion in Ménière's disease is within the auditory nerve itself. The disease is almost exclusively one of middle or more advanced age, and, for reasons unknown, the left auditory nerve is the site of the attacks about twice as frequently as is the right. Ménière's disease can be permanently cured by division of the auditory nerve. Partial section spares the hearing and in most cases is just as effective in curing the attacks. Tinnitus is abolished in about 50 per cent of cases. The procedure is carried out with local anesthesia. There was 1 death, due to meningitis, in 401 operations which the author performed. No disturbances except for temporary dizziness are noted after operation. Bilateral section produces jumbling of objects (visual) when the patient is in motion and uncertainty in walking in the dark, but no disturbance in gait. The Romberg sign is not present. The results are satisfactory and permanent.

HENRY A. MILLER, Philadelphia.

RECONSTRUCTION OF THE EAR. J. NEWMAN, Surg., Gynec. & Obst. **73**:234 (Aug.) 1941.

In the past a reconstructed ear was clumsy and bulky because an excessively thick tubed-pedicle was used for the helix. Thin tubes can be made to function if the pedicle is delayed by undercutting and resuturing. Using the general plan of G. W. Pierce, two ears were reconstructed. The cosmetic results were excellent. General anesthesia was employed when the cartilage was removed; otherwise the remaining eight operations were performed with local anesthesia. Each patient spent an aggregate of eighteen days in the hospital.

HENRY A. MILLER, Philadelphia.

ORGANIZATION OF INSTRUCTION IN SCHOOLS FOR DEAF-MUTES. F. CHAVANNE, Oto-rhino-laryng. internat. **24**:289 (Dec.) 1940.

Chavanne criticizes the present practice of segregating the deafened in special schools, where they are isolated from normal children and from life in general. Condemned also is the practice of grouping deaf-mutes of normal intelligence with backward children or those with subnormal mentality. The mentally subnormal children should be placed in special institutions. The instructors should be chosen from those who have had experience in teaching the deafened and should be adequately compensated. Directors should be chosen from among the instructors.

DENNIS, San Diego, Calif.

VITAMIN B_1 THERAPY FOR NERVE DEAFNESS. SEIICHI KAWATA, Oto-rhino-laryng. **13**:881 (Dec.) 1940.

Kawata records the cases of 30 patients with perceptive deafness (of unknown origin) who were treated at the university clinic at Fukuoka, Japan. Seventeen were males and 13 females. Cases of deafness resulting from the use of drugs, cases of nerve deafness of syphilitic origin and cases in which there was evidence of Ménière's syndrome were not included. In the majority the onset was before the age of 20. All but 1 patient had bilateral involvement. Sixteen complained of tinnitus. Twelve (40 per cent) had a history of family deafness or nervous instability. None showed tubal obstruction, and all gave a positive reaction to the Rinne test. For the treatment thiamine hydrochloride was given by mouth as well as subcutaneously in massive doses for a month or longer. Twenty-two patients (73.3 per cent) improved in hearing, and tinnitus was much less troublesome or was completely eradicated.

HARA, Los Angeles.

Pharynx

FRESH FOREIGN BODY LESION OF THE ESOPHAGUS COMBINED WITH AN OLD FOREIGN BODY HEALED INTO THE THYROID; MISLEADING DIAGNOSIS; CAUSATIVE CONNECTION. H. WULLSTEIN, Monatschr. f. Ohrenh. **75**:82 (Feb.) 1941.

In a patient complaining of difficulty in swallowing, a fresh lesion of the esophagus was noted. Roentgenographic examination revealed the presence of a needle, located outside the esophagus and certainly not in the region of the fresh lesion. It was found that a goiter had dislocated and partly obstructed the esophagus, and it is assumed that the needle had perforated the esophageal wall years ago and entered the thyroid without causing any symptoms.

The thyroid capsule is held responsible for having hindered the further wandering of the foreign body. The second foreign body, probably a bony spicule, recently caused a superficial lesion of the mucosa and passed on. As this happened in the same region, although not in the same spot, it can be assumed that the esophagus was slightly compressed or dislodged in this region and, therefore, was more apt to stop foreign bodies at this point.

LEDERER, Chicago.

ERYSIPELAS OF THE ORAL MUCOSA FOLLOWING TONSILLECTOMY. W. SPRENGER, Monatschr. f. Ohrenh. **75**:87 (Feb.) 1941.

Among 2,290 cases of tonsillectomy at the Innsbruck clinic, 10 cases of erysipelas were encountered. All of the patients were cured under treatment with "prontosil" (preparation used not specified), without major complications. It is questionable whether the erysipelas was due to the operation directly, as it appeared only two to three days after; so a secondary infection (negligence by the patient in eating raw fruit) was more likely. A specimen for biopsy was taken from the posterior pillar of one of the patients. It showed a mucosa with stratified squamous epithelium covered by a thin horny layer. All of the mucosa was thickly infiltrated with leukocytes; the thin horny layer was elevated in the form of small vesicles containing pus. There was edema of the connective tissue; no bacteria were present. This picture conforms with the findings of Dietrich, who described erysipelas of the oral mucosa as a serous inflammation. An illustrative photomicrograph accompanies the article.

LEDERER, Chicago.

THERAPEUTIC EFFECT OF TONSILLECTOMY ON RHEUMATISM. FRANTIŠEK KOTYZA, Časop. lék. česk. **76**:1681 (Oct. 8) 1937.

This report is based on 2,458 tonsillectomies from Přecechtěl's clinic. Rheumatism was the indication in 1,140 cases, or 46 per cent. There were a history of familial rheumatism in 172 and a history of parental tuberculosis in 6 per cent

of the cases. The prodromal diseases, in the order of frequency, were measles, scarlet fever and diphtheria. The sex incidence was: females, 58.3 per cent; males, 41.7 per cent. The majority of the patients had acute or repeated tonsillitis before the operation. Rheumatic attacks antedated acute tonsillitis in 54 per cent of the cases, and in most of the others rheumatism was present during the attacks of tonsillitis. Postoperative articular involvement took place in 4.8 per cent and inflammation of the joints in 1.2 per cent. There was rarely definite improvement of acute articular symptoms, though the best results were obtained when the operation was performed within a year after the onset of articular symptoms. Of a control group of 100 cases, improvement was noted in 94. Active endocarditis was considered a contraindication to any operative procedure.

STULIK, Chicago. [AM. J. DIS. CHILD.]

Larynx

PRESENT STATUS IN THE TREATMENT OF ACUTE OBSTRUCTIVE INFECTIONS OF THE LARYNX, TRACHEA AND BRONCHI. H. R. LITCHFIELD, Arch. Pediat. **57**:717 (Nov.) 1940.

Twenty-nine cases are reported; only 2 deaths occurred. Fifty per cent of the patients were infants. Cultures of material from the throat yielded a variety of organisms, including Streptococcus haemolyticus, Micrococcus catarrhalis, Staphylococus aureus and Haemophilus influenzae.

Treatment consisted of laryngoscopic and bronchoscopic examinations, with removal of mucus, and, when the infection was severe, tracheotomy. Further treatment consisted of restoration of fluids, inhalations of steam and administration of oxygen.

ORR, Buffalo. [AM. J. DIS. CHILD].

NEW ARTIFICIAL LARYNX. W. L. HANSON, Illinois M. J. **78**:483 (Dec.) 1940.

Dr. Hanson presents an instrument devised by a laryngectomized patient who was unable to educate himself to a satisfactory buccal-pharyngeal or esophageal voice. The general plan is the same as that of the other instruments on the market. However, the author claims the following improvements: The instrument is made of 98 per cent hard rubber, which eliminates metallic sounds and produces a tone closer to that of the natural human voice. The lay tapers to a hair line edge and requires less air pressure. Saliva is prevented from holding the reed by capillary attraction, which thus is free to act more of the time. Less and more uniform pressures are also required in the use of this tube.

This artificial larynx is one-half the weight of the others on the market. It is more resistant to condensation of moisture and has only one movable part, which has a breather valve hidden from view, thus greatly relieving manual operation of the thumb over the breather hole. The instrument is easily cleaned. It will not bend or break if dropped.

ROBERT B. LEWY, Chicago.

POSTDIPHTHERITIC STENOSIS OF THE ESOPHAGUS. A. FRONIME, Beitr. z. klin. Chir. **168**:604, 1938.

Less than a dozen cases of this condition have been reported in the literature in the last one hundred years. In the majority of the cases in which such complications occur the patient dies because of the severity of the infection.

The case reported is that of a 22 year old patient who contracted diphtheria at 6 years and again at 20 years of age. There was gradually increasing difficulty in swallowing. Interesting roentgenograms taken before and after operation are presented.

RUBIN, Boston. [AM. J. DIS. CHILD.]

PERMANENT BOUGIE IN THE TREATMENT OF ACUTE CORROSIVE ESOPHAGITIS. S. BELINOFF, Monatschr. f. Ohrenh. **74**:587 (Dec.) 1940.

Clinically, three forms of acute corrosive esophagitis can be distinguished—slight, mild and severe. The slight form (10 per cent incidence), in which only a little poison gets into the esophagus, requires no treatment. The severe form (27 per cent incidence) is to be treated only with palliative measures, as death ensues in a short time. The third form (63 per cent incidence) is suitable for treatment. The author believes that in all cases of acute esophageal burns esophagoscopy is indicated and is of no danger if executed properly. Early blind bougie treatment, performed without previous intraesophageal examination as to the localization and severity of the lesion, may prove very dangerous. The present day treatment by daily dilatation with hard bougies (Bouchard) and Salzer's method are not advisable, as the damaged membrane is daily irritated anew. A permanent bougie, as advised by Gersuny sixty years ago, can be brought in position by means of external pharyngotomy and can remain in place for weeks. This was done by the author in 5 cases, with excellent results. No reflexes are prompted, as the pharynx is eliminated; feedings can be given through the bougie, and good drainage is provided thereby to obviate possible periesophagitis and mediastinitis.

LEDERER, Chicago.

ENDOTHELIOMA OF THE LARYNX. TADASHI WATANABE, Oto-rhino-laryng. **13**:991 (Dec.) 1940.

Endothelioma involving the larynx is comparatively rare. Manasse recorded 4 cases. Harne, Jackson, Nourse, Lambert, Lock, Hino, Fujii, Kubo and Kikkawa each reported 1 or more cases.

Watanabe reports a case of a Korean married woman aged 41 years. Her chief complaints were hoarseness and dyspnea. Thirteen years before admission to the university clinic, during the eighth month of her second pregnancy, huskiness of voice and dyspnea developed without apparent cause. The condition became progressively worse, and at the time of her confinement she was aphonic and extremely dyspneic. After delivery her symptoms gradually improved, without any medication. During her third and fourth pregnancies there was recurrence of symptoms, with similar improvement after each confinement. There was no dysphagia. No relation between menstruation and the severity of symptoms was apparent. Her condition was worse during the winter than during the summer months. Laryngeal examination revealed that the growth was somewhat oval and filled the false and true cords and the glottis, leaving only a small portion of the air passage free at the posterior commissure. Biopsy revealed lymphendothelioma. The relation of pregnancy to the growth of the tumor is not altogether clear.

HARA, Los Angeles.

Nose

CORRECTIVE SURGERY OF THE NASAL TIP. JOHN MARQUIS CONVERSE, Ann. Otol., Rhin. & Laryng. **49**:895 (Dec.) 1940.

The anatomy of the nasal tip is discussed, with consideration of the development. The author then discusses the diagnosis of the types of deformities and the various surgical procedures used in their correction. Numerous diagrams and photographs accompany the article.

M. V. MILLER, Philadelphia.

NASAL ATRESIAS: A SURGICAL CRITIQUE. ALBERT A. CINELLI, Ann. Otol., Rhin. & Laryng. **49**:912 (Dec.) 1940.

Atresia of the anterior nares, the choanae and the nasopharynx is discussed, and the literature is reviewed. Short descriptions of various technics of repair and correction are given, and the bibliography appears full.

M. V. MILLER, Philadelphia.

OSTEOMA OF THE FRONTAL SINUS. G. SALVADORI, Arch. ital di otol. **52**:539 (Nov.) 1940.

In 1938 Malan collected 458 cases of osteoma of the perifacial cavities, in 178 of which the tumor was in the frontal sinus, in 110 in the ethmoid sinus, in 41 in the maxillary sinus and in 7 in the sphenoid sinus.

Osteoma must be differentiated from exostoses, hyperostoses and osteophytes. Salvadori reports a case in a girl in which the tumor involved the lateral half of the right frontal sinus and the orbit and penetrated the external wall of the sinus. The tumor was easily shelled out, and no attachment to the underlying bone was observed. Histologically the tumor was a spongy osteoma. The theories of the pathogenesis of osteoma are discussed. The author concludes that the tumor was a free osteoma, originating in the anterior pole of the ethmoid labyrinth, extending laterally and downward and penetrating the outer wall. The pedicle had been lost. The probable source of the tumor was an embryonal cartilaginous rest. Osteomas occupy a place between benign and malignant tumors. Clinically they owe their gravity to possible orbital or intracranial complications. Since the inner wall of the sinus and the nasofrontal duct were normal, no enlargement of the latter was made. The patient made a prompt and lasting recovery.

DENNIS, San Diego, Calif.

STRUCTURE OF THE VESSELS IN THE NASAL MUCOSA OF THE RESPIRATORY REGION. C. GUARAGNA, Arch. ital. di otol. **52**:615 (Dec.) 1940.

The difference between the cavernous bodies of the nose and true cavernous tissue is that in the former the blood is carried by the veins and not by the arteries. In the nose afferent arterioles have never been observed, and the veins have a rich muscle supply. In proportion to the great quantity of veins, the arteries are few and of minor caliber. Even before they enter into relation with the mucosa they give off periosteal branches that are resolved in the mucosa into a fine capillary network, which latter empties into venules that open into the deep layer of venous plexuses or into the trunks of efferent veins.

The material for Guaragna's studies was the mucosa from the margin of the inferior turbinate, the floor of the nose and the locus Valsalvae from the cadavers of 7 adults, whose ages varied from 27 to 60 years. The most interesting observation was the discovery, in the arteries with a longitudinal course in the periosteum, of raised folds, analogous to those observed by Kiss in the arteries of the corpus cavernosus of the penis. These folds are not to be confused with thickening of the intima due to senile modifications, described by Saxen. Also, in senile modifications the elastic fibers appear broken and softened. Such changes are observed only at a more advanced age and were not seen in the material studied. Specimens of mucosa from the different regions showed in general the same disposition of the vessels, varying only in degree. On the basis of the anatomic data, a different manner of functioning of the cavernous structures of the nose from that of the penis may be postulated. The existence in the veins of a proper wall with a muscular tunic is confirmed, and its relation with the bundles of elastic fibers of the lamina propria of the mucosa is demonstrated.

DENNIS, San Diego, Calif.

INDICATION AND DANGERS OF INTRANASAL EXPOSURE OF THE FRONTAL SINUS. Z. TATRALLYAY. Monatschr. f. Ohrenh. **74**:626 (Dec.) 1940.

The operative technics of Halle, Jansen, Killian and others on the frontal sinus contain too many dangerous elements, so that the author was inclined to try another approach in cleaning up chronically diseased frontal sinuses. According to his experience, the mucous membrane of the frontal sinus will return to normal when sufficient drainage is achieved. This was carried out in 31 cases by removing the agger nasi cell, without any complications. The agger nasi and adjacent anterior ethmoid cells were removed through the endonasal approach, and in all cases the

condition cleared up in due time. The author lays special stress on the fact that in this approach the danger of osteomyelitis is eliminated, a complication which had previously cost the life of 1 of his patients. LEDERER, Chicago.

Miscellaneous

THE ANTI-ENZYMATIC NATURE OF SULPHANILAMIDE'S BACTERIOSTATIC ACTION. R. MELLON, A. LOCKE and L. SKINNER, Am. J. M. Sc. **199**:749 (June) 1940.

The authors present an antienzymatic approach to the modus operandi of sulfanilamide. Blood satisfies one of the requisites of a good bacterial culture medium in that it contains substances that neutralize the waste products formed by the growth of the micro-organisms. One of these substances, the enzyme catalase, destroys the growth-checking hydrogen peroxide normally formed by hemolytic streptococci and pneumococci, not permitting it to accumulate to levels which would be toxic to the organism. Experimental evidence indicates that when sulfanilamide undergoes slight oxidation it becomes anticatalytic. The free amino group of sulfanilamide after incomplete oxidation inactivates catalase, thereby allowing accumulation of hydrogen peroxide in quantities toxic to the germ. This reduces its growth rate and renders it vulnerable to the immune forces of the blood stream, which complete its destruction. Experimentally it was shown that detectable peroxide always appeared in from two to three hours in both sulfanilamide and control cultures of pneumococci and that from the third hour retardation in growth was apparent in the cultures containing sulfanilamide.

When aqueous solutions of sulfanilamide are exposed for a brief period to ultraviolet light, the anticatalytic property is enhanced. The experimental evidence suggests that the oxidative processes going on within the growing organism represent the oxidative equivalent of ultraviolet light. It could be demonstrated that a progressive decrease in oxygen tension of the culture is accompanied by a decrease in the bacteriostatic effect of sulfanilamide.

In addition to catalase, enzymes adversely affected by the intermediate oxidation products of sulfanilamide and its compounds are peroxidase, nitrotase and certain dehydrogenases. SHAMBAUGH JR. and JENNES, Chicago.

THE RÔLE OF WATER BALANCE IN THE CLINICAL MANIFESTATIONS OF ALLERGY. R. A. KERN, Am. J. M. Sc. **199**:778 (June) 1940.

In a convincing manner, the author suggests the theory that changes in the water balance of the body may influence the occurrence of allergic phenomena. A basic component of the allergic reaction is edema in the shock organ, and the production of this is quantitative. If for a given patient the figure 100 represents the amount of allergenic action required to produce shock organ edema and consequent symptoms, then if the patient is subjected to an allergenic action of a magnitude of 95 no edema and no symptoms result, whereas an allergenic action in an amount of 105 produces edema and symptoms. In allergic patients symptoms may be the result of the concerted action of two causes of edema, the one (allergenic) acting locally in the shock organ, the other a cause of generalized hydration. The important one with respect to magnitude of local effect is the allergenic factor, whereas less important as to magnitude, but at times the only obvious factor, may be any one of several causes of water retention. Large values are assigned to the allergenic action for producing edema, as the effect is concentrated in one small structure, the shock organ, whereas in the case of other factors influencing edema, though a much greater actual amount of water is involved, the shock organ participates to no greater degree than does any other tissue and therefore comparatively small values are assigned to them.

In the case of a patient subjected to an allergenic action of 95, insufficient in itself to produce allergic edema, edema will develop in the shock organ if, in addition, he is subjected to another type of edema producer in the magnitude of,

let us say, 10. This, then, explains why patients may show allergic symptoms on a day when a severe storm is approaching, for low barometric pressure favors retention of body fluids; similarly with the menses, for menstrual edema is well recognized.

Conversely, the patient who is experiencing edema of his shock organ and consequent symptoms due to an allergenic action of the magnitude of 105 will experience regression of shock organ edema and symptoms when an antiedema factor of a magnitude of 10 comes into play. This antiedema factor might be the dehydration of diabetes, of acidosis or of administration of potassium chloride.

Sodium, which occurs for the most part extracellularly, influences the amount of interstitial fluids, whereas the administration of potassium, which is primarily intracellular, produces first an increase in intracellular water at the expense of interstitial water. This, then, is the rationale of administration of potassium chloride and the restriction of sodium chloride in cases of allergy. Causes initiating changes in water balance must not have attributed to them any specific etiologic significance in the causation of the allergy itself.

SHAMBAUGH JR. and JENNES, Chicago.

THE USE OF SULFAPYRIDINE IN STREPTOCOCCUS VIRIDANS MENINGITIS. W. J. MITCHELL, A. BOWER and P. HAMILTON, Am. J. M. Sc. **200**:75 (July) 1940.

The authors have recently employed chemotherapy in 6 cases of meningitis caused by Streptococcus viridans. Two patients were treated with sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) and recovered. One was treated with sulfanilamide and sulfapyridine and finally with N, N-dimethyl-N^4-sulfanilylsulfanilamide and high voltage roentgen therapy and recovered. Three were treated with sulfanilamide and failed to improve. The authors conclude that sulfapyridine was responsible for the recovery of 2 of these patients and had an inhibiting effect on the organisms in the third.

SHAMBAUGH JR. and JENNES, Chicago.

MAXILLOFACIAL INJURIES: IMPORTANT CONSIDERATIONS. JAMES J. IVORY, Indust. Med. **10**:52 (Feb.) 1941.

The final result in the treatment of maxillofacial injuries is dependent on early recognition and efficient treatment. Close collaboration with the oral surgeon is of the utmost importance, as he can usually detect fractures of the jaws and facial bones by inspection and palpation; and this is of particular aid when adequate roentgenologic examination cannot be made because of the general condition of the patient.

The control of hemorrhage, the establishment of an unobstructed airway and the relief of pain constitute the early routine. Surgical cleansing to remove any foreign particles is the first step before immobilization, as infection is the complication which stands out in these cases.

The object of treatment is to correct the facial deformity, establish normal occlusion of the teeth and maintain the fragments in their proper apposition until union occurs. Usually, perfect anatomic position means a perfect functional result.

If teeth are present, intermaxillary wiring of the teeth in occlusion is the preferred method. If teeth are not present, circumferential wiring of the mandible over a splint or a denture is necessary. Depressed malar fractures produce deformity, cause interference with mastication and may be associated with visual and ocular disturbances. Correction is best accomplished by elevating the fragments with a tenaculum introduced through the soft tissues.

H. A. MILLER, Philadelphia.

REVIEW OF 3,000 OPERATIONS ON OUTPATIENTS PERFORMED IN THE OFFICE. W. HAARDT, Monatschr. f. Ohrenh. **74**:638 (Dec.) 1940.

More than 3,000 operations, ranging from paracentesis to radical mastoidectomy and from polypectomy to sinus operations, were performed by the author

without any untoward incident or complication. General advice is given regarding the care of the patients and the management of their relatives. Special attention is given to the problem of tonsillectomy on ambulatory patients; the possible bleeding points are discussed, and the necessity of control of bleeding is stressed.

LEDERER, Chicago.

EXPERIENCES WITH THE EMPLOYMENT AND DOSAGE OF SCOPOLAMINE–DIHYDROOXYCODEINONE HYDROCHLORIDE (EUKODAL EPHETONINE) IN THE PRACTICE OF OTORHINOLARYNGOLOGY. H. SCHNAUBELT, Monatschr. f. Ohrenh. **75**:69 (Feb.) 1941.

The intravenous use of a combination of scopolamine hydrobromide (0.0005 Gm.), dihydroxycodeine hydrochloride (eukodal) (0.01 Gm.) and ephetonine (0.025 Gm.) to aid in the induction of local anesthesia is advocated. From 1 to 1.5 cc., according to the age and weight of the patient, is given thirty minutes before operation. The patient will be semicomatose but able to obey orders and will have no recollection of the events. A number of cases (134) in which anesthesia was obtained by this method are reported.

The operative procedures in these cases included radical sinus operation, removal of a peritonsillar abscess, tonsillectomy (30 cases) and tonsillectomy performed with the patient under local anesthesia.

LEDERER, Chicago.

Society Transactions

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

JOHN MACKENZIE BROWN, M.D., *President*

E. W. HAGENS, M.D., *Editor of Abstracts*

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Tumors of the Larynx Other Than Squamous Cell Epithelioma. DR. FRED Z. HAVENS and DR. EDITH M. PARKHILL (by invitation), Rochester, Minn.

This paper was published in the December 1941 issue of the ARCHIVES, page 1113.

DISCUSSION

DR. LOUIS H. CLERF, Philadelphia: My experience with malignant tumors of the larynx other than squamous cell carcinoma has been limited to 8 cases, during a period when slightly more than 650 cases of squamous cell carcinoma were observed. These 8 cases constitute about 1 per cent of all cases of malignant tumors of the larynx encountered in my experience. The tumors comprised 1 adenocarcinoma, 2 hemangioendotheliomas and 5 sarcomas, all but 1 of which occurred in men.

Practically all of the nonsquamous cell types of malignant tumors exhibit benign characteristics early in their course. When once the growth is discovered it should be investigated, so that an accurate diagnosis may be made and appropriate treatment instituted. In addition, one should always regard cases of chondroma with suspicion and should employ only those measures that have for their purpose complete removal of the growth.

DR. H. B. GRAHAM, San Francisco: I examined the records of the past thirty years of the Stanford University Hospitals, the San Francisco Hospital and the Southern Pacific General Hospital and in my own private practice, and I found at the Stanford University Hospital but 1 bona fide case of adenocarcinoma and another in which the growth reacted favorably to combined treatment, and which I excluded for lack of knowlege of the end results, and 1 case of chondrosarcoma occurring in my private practice.

DR. THOMAS E. CARMODY, Denver: My associates and I have found two types of carcinoma in one tumor. I think that is more important than finding two different tumors; I wish Dr. Havens would enlarge on that.

DR. FRED Z. HAVENS, Rochester, Minn.: I should like to reemphasize the point I first made, that one must be very careful in the diagnosis of chondrosarcoma. One must be sure of the pathologist. One must look thoroughly for evidence of malignancy. We lost 1 patient in our group whose sarcoma was due to failure to recognize the malignant nature of the growth.

As I said, we do encounter occasional instances in which there are two types of malignant growth. The same thing is observed in some lesions of the skin. There is a basal epithelioma, and mixed with it is some squamous cell epithelioma. There are two types of cell changes, both of which are malignant. The same is true of mixed tumors of the larynx; one finds changes in both directions. The adenomatous structure undergoes malignant changes and gives rise to an adenocarcinoma, and some of the cells change into squamous cell epithelioma, so that both types of carcinoma occur in the same tumor.

Modified Combination of Irradiation and Surgical Therapy in Treatment of Late Cancer of the Throat, with Reports of Cases. Dr. Millard F. Arbuckle and Dr. A. C. Stutsman, St. Louis.

It is now twelve months since we began treating 7 patients by this method. The results during and after treatment have been so worth while that we think a further report is justified. The patients were all in the late stages of laryngopharyngeal cancer when first seen. Glandular involvement was uniformly present, and in 1 case cancer involved both lobes of the thyroid. The diagnosis was confirmed microscopically in each instance, and an effort at grading was made. One woman in the group had cancer of the cricoid cartilage. The patients were all of middle age or more, and several were aged.

The procedure is carried out under anesthesia induced by avertin with amylene hydrate, supplemented with local anesthesia. A midline incision is made and a tracheotomy tube is inserted. The thyroid cartilage is skeletonized and divided in the midline. The perichondrium is elevated from the internal and external surfaces of the thyroid cartilage, and each half is completely removed, including the superior cornu. The epiglottis, if involved, is approached through the thyrohyoid membrane. It is pulled down into the wound and amputated. In several cases we did a submucous resection of the epiglottic cartilage. Bleeding may be troublesome in those cases in which the epiglottis is involved with the tumor. The wound is closed in the usual way, and a nasal tube is inserted. In eight to twelve days after operation, depending on the reaction of the patient, fractionated irradiation is started.

In conclusion, it is important to remember that this method is suggested only in those cases in which an extrinsic carcinoma of the larynx is present, that is, carcinoma of the epiglottis, of the hypopharynx or of the cricoid cartilage. We are aware that in the present cases the post-treatment period is not long enough to permit judgment of the final results, but up to the present we have been tremendously impressed with the increased comfort of the patient and with the effect on the carcinoma.

DISCUSSION

Dr. Edward Whalen, Hartford, Conn.: Malignant neoplasms of the pharynx are beyond the scope of surgery because of their growth in loose connective tissue and, too, because of early lymphatic spread. These tumors all show but little cell differentiation, which takes them out of the operable group but does qualify them for radiation therapy. The method used by the authors gives the roentgen therapeutist a free hand in that it removes the specter of radionecrosis of cartilage, a reality that has done much to hamper the roentgenologist in his treatment of the cervical region. The authors have made use of planigraphic studies of these larynxes. By means of laminagrams they have determined the extent of the lesion and whether calcification of the cartilage has taken place, and they have tried to estimate the amount of invasion of this tissue by cancer.

Dr. William E. Costolow, Los Angeles: Removal of a cartilage before irradiation brings up several problems. First, there is the danger of operating in a field which may be involved with cancer or of the field's becoming infiltrated with cancer. Moreover, how is one to know, when removing these cartilages, whether or not they may be involved with cancer? Certainly, if they are, one will not help matters much. Second, experience in irradiation of other parts of the body has taught that tolerance to radiation is definitely lessened by a previous surgical procedure. With the present technics, whereby small fields, usually not larger than 7 cm. in diameter, are used, damage to cartilage may be kept at a minimum; in fact, in the majority of cases it may be almost entirely avoided. From the standpoint of irradiation I cannot see the logic of removing the cartilages. Certainly, one will not be able to use any more adequate irradiation than is now possible.

Dr. M. C. Myerson, New York: This paper is a real contribution, but it must be added that the method is applicable to a very small, selected group of patients. I think it is timely that a policy of more radical and extensive surgical

intervention in this field be adopted. Once the epiglottis is involved it is best to remove it.

DR. H. B. GRAHAM, San Francisco: I have been watching for a report from Dr. Portmann on his original operation, but I have seen none. I was enthusiastic about this operation at the time he first described it and used it in 1 of my cases. I have never seen such rapid infiltration of tissues by a carcinoma as occurred in that case. It may have been that the roentgen application was insufficient, but I have never since attempted a resection of the cartilage of the larynx.

DR. LOUIS H. CLERF, Philadelphia: I have found that the best results probably are secured not by irradiation alone but by radical surgical intervention followed by irradiation. When I say "radical" I want it interpreted literally, for I feel that the patient will do far better by having the larynx removed. If one is going to take out the cartilage, one will probably have to take out the epiglottis, the thyroid and the cricoid cartilage. This is likely to cause trouble because there will be more necrosis; so while one is operating one had better take out the larynx. Many roentgenologists say these large carcinomas do not respond well, and that has been my experience.

DR. STEWART HARRISON, Los Angeles: Dr. Portmann abandoned his method and afterward used roentgen rays.

DR. HAROLD A. FLETCHER, San Francisco: I feel very strongly on one point. If one is going to combine surgery and irradiation in these cases, one should operate first and irradiate afterward. Roentgen treatment destroys most of the hope of good healing for surgical procedures which may follow.

DR. A. C. STUTSMAN, St. Louis: The only purpose of this procedure has been to save the patient the great suffering due to the presence of dead cartilage. We have not seen any spread of the carcinoma from the operative procedure we have employed. As far as radical operation is concerned, we have been removing large masses of carcinoma at the time the cartilage is removed.

Carcinoma of the Larynx in a Girl Aged Fifteen. DR. ARTHUR C. JONES, Boise, Idaho.

This article was published in the March 1942 issue of the ARCHIVES, page 426.

DISCUSSION

DR. CHEVALIER L. JACKSON, Philadelphia: I believe that this is an important contribution because it puts on record an unusual case of cancer. I have not seen a younger patient with cancer of the larynx, and I do not recall having found a case of one so young in the literature.

DR. H. B. GRAHAM, San Francisco: Some years ago I had a patient under 18 years of age whose lesion was diagnosed microscopically as carcinoma of the larynx. I was much surprised when Dr. Clerf said he had not seen such a condition in a patient under 19 years of age and when Dr. Jackson stated he had never seen a case in children.

DR. FRED Z. HAVENS, Rochester, Minn.: The management of carcinoma of the larynx is an important problem to bear in mind. The men who are dealing with cancer work in teams, and teamwork is important in the proper management of this condition.

Interrelations, Diagnosis and Treatment of Benign and Malignant Tumors of the Larynx, with Special Reference to the Prevention and Cure of Laryngeal Cancer. DR. GABRIEL TUCKER, Philadelphia.

Observations on a fairly comprehensive series of cases of benign and malignant lesions of the larynx occurring in my own experience have led to the conclusion that these tumors have many points in common, such as site of the lesion and common etiologic factors of local irritation by functional trauma, external irritation and the use of alcohol and tobacco. The normal transitional changes which

occur in the mucous membrane of the larynx as a result of advancing age would supply the histologic basis for a benign lesion's becoming malignant in patients who have the unknown etiologic factor of predisposition to the development of cancer. The treatment of a benign lesion by removal and the restoration of the affected area to normal is a well established and logical procedure. The treatment of a malignant lesion of the larynx is also fairly well standardized. The majority of laryngologists believe that laryngofissures should be performed in cases of early cancer of the cord and that laryngectomy is the treatment of choice for more advanced lesions involving the vocal area and adjacent parts. The rehabilitation of the patient, acquisition of a voice after laryngofissure or laryngectomy, is fairly satisfactory. Laryngectomy by an operative procedure in which the muscles are allowed to remain in the neck is believed to favor the development of a satisfactory esophageal voice.

As a preventive measure in the development of cancer, it is believed that the removal of benign tumors, particularly of the vocal cords, and the restoration of the contour and function of the cord may be an important factor in the prevention of cancer. Histologic examination of the tissue removed by direct laryngoscopy makes possible the diagnosis of the cancer at the earliest possible time, so that surgical treatment may be more effectively carried out.

Irradiation after operation in cases of extensive lesions and in cases of postoperative recurrence has been found of great value. It is particularly effective in cases of local recurrence and of cancer in persons over 65 years of age. Extrinsic cancer in which metastatic extension occurs early is best treated by irradiation in most cases.

DISCUSSION

DR. WILLIAM H. JOHNSTON, Santa Barbara, Calif.: I believe that practically all growths on the vocal cords should be considered malignant until proved otherwise. I recently had the experience of making a biopsy in a case in which almost the entire growth was removed and appeared to be benign to the naked eye. The patient was told that in all probability no further treatment would be necessary. A few days later the pathologist reported that the growth was malignant.

Dr. Tucker brings a thought which is new to me and most interesting, that is, that the upset of the normal mechanism of voice production by a benign growth may result in recurrence and possibly in production of a malignant growth if reeducation of the laryngeal musculature is not carried out. I believe with Dr. Tucker that there is no excuse for not removing a benign growth and restoring normal function to the vocal cords. The recent paper by Dr. Broyles in which he reported that the use of estrogen seems to have reduced the multiplying and spreading characteristics of papilloma in children is of special interest, and the treatment deserves trial. Twenty-five or more years ago many laryngologists advised strongly against biopsy of new growths in the larynx. I believe that quite the reverse is true today.

Stress should be put on the periodic observation of patients who had a benign growth removed from the larynx. Two points cannot be too often emphasized: 1. Every patient who has persistent hoarseness should have a careful examination of the larynx by a competent laryngologist. 2. Histologic examination of the tissue removed for biopsy will make possible the diagnosis of laryngeal cancer at the earliest possible time, so that surgical treatment can be effectually carried out.

DR. GABRIEL TUCKER, Philadelphia: Unquestionably, a great many men have the feeling that a benign lesion may go on for years and later become malignant. This is true of papilloma in adults.

Calculus of the Salivary Glands. DR. EMIL THOLEN, Los Angeles.

This paper will appear in a later issue of the ARCHIVES.

DISCUSSION

DR. J. B. NAFTZGER, Hollywood, Calif.: The rather meager literature on salivary calculus has brought out little new with regard to its origin. Most

observers believe that calculus is due to inflammation of the salivary gland, with chemical changes in the secretions, to bacteria or a foreign body acting as a nidus for the formation of concrement or to faulty calcium metabolism. Considerable emphasis is placed by some investigators on dental caries as a possible exciting cause. However, Karsham and associates, in 1930, in a study of the chemical changes in the saliva with and without dental caries, found that calculi developed in inverse proportion to the amount of dental caries. The more frequent involvement of the submaxillary gland (about 70 per cent) would point to the infection theory because of the greater accessibility of the submaxillary duct.

As Dr. Tholen states, the majority of cases appear in later life, although there is wide diversity in the ages. Harrison reported 1 case in which a stone was found in the lingual duct of a baby 3 weeks old.

In diagnosis, it must be remembered that there may be remissions of the inflammatory symptoms over long periods and that eventually the calculus may come forward into the duct. Sometimes the stone sloughs through the duct and is extruded spontaneously. It seems wise, therefore, not to be hasty in operating on the submaxillary gland unless serious symptoms occur. In my experience the great majority of calculi can be removed through the mouth.

Dr. Thomas E. Carmody, Denver: Dr. Tholen has said that the submaxillary gland is most often affected but that the parotid gland may be affected also. I have seen a number of cases of parotid involvement over a period of years. One patient has an attack of double parotitis every six months. I have been unable, even with iodized poppyseed oil, to find any calculi in the ducts. The only thing that has given him relief is to break down the inflammatory reaction. He will then get along for another six or eight months without any trouble.

Dr. James G. Dwyer, New York: I think that calculi are often overlooked. In twenty-eight years I have had 24 cases. Another point to remember is that these calculi, like kidney stones, do not always show in the roentgenogram. One may have a stone and not know it.

Primary Pseudocholesteatoma of the Ear. Dr. Kenneth M. Day, Pittsburgh.

This article was published in the December 1941 issue of the Archives, page 1144.

DISCUSSION

Dr. Rea Ashley, San Francisco: The classification of cholesteatomas as true and false, with subdivision of the pseudocholesteatomas into secondary and primary types, depending on the presence or absence of infection, is excellent and makes clear a hitherto confused terminology.

Dr. Pierre Viole, Los Angeles: Dr. Day's paper on primary pseudocholesteatoma of the ear, with particular reference to its causation in the improper feeding of infants, is interesting and thought provoking. The term pseudocholesteatoma would seem to answer the question: When is a cholesteatoma not a cholesteatoma?

Like many others, I have divided cholesteatomas into only two types: the congenital, or true, cholesteatoma, which, incidentally, I have never personally encountered in the temporal bone, and the cholesteatoma that is secondary to a foreign irritation, regardless of the presence or absence of suppuration.

Dr. Frank B. Kistner, Portland, Ore.: I wish to speak principally of cholesteatoma of the sinuses. Many men deny that there is such a thing, but I have seen 4 cases.

Dr. Kenneth M. Day, Pittsburgh: As to the fluid entering the eustachian tube when the infant is feeding: If the baby is turned on its side some of this can be avoided. A baby cannot swallow properly when lying on its back. I tried that myself and found that one cannot swallow that way. There is a tendency to force the food into the nasopharynx. By rolling over on the side and elevating the head one can swallow.

Treatment and Prognosis of Aural Infections Complicated by Severe Diabetes. Dr. Alice G. Hildebrand (by invitation), Dr. Bert E. Hempstead and Dr. Edwin J. Kepler (by invitation), Rochester, Minn.

The series of patients in this study consisted of 5 men and 6 women. Their ages ranged from 19 to 70 years and averaged 48 years; 72 per cent of them were 40 years of age or more.

All of the patients had unilateral acute or chronic suppurative otitis media and acute or chronic mastoiditis. At operation associated petrositis was observed in 6 patients, sigmoid or lateral thrombophlebitis in 2, extensive cellulitis of the neck in 1 and extensive osteomyelitis of the temporal bone in 1. Five of the 10 patients subjected to operation made uneventful recoveries. Three who were operated on died; the operative mortality rate was therefore 30 per cent.

Sulfanilamide and its derivatives must be used with discretion. Many of these compounds produce nausea and vomiting, both of which are highly undesirable complications of diabetes. Other things being equal, therefore, the drug which produces the least gastrointestinal disturbance is the one of choice.

Conclusions.—1. Aural infection occurring among patients suffering from diabetes should be regarded from the first as a serious illness with dangerous potentialities.

2. Infection intensifies diabetes, and poorly controlled diabetes leads to further infection. For this reason, the patient who has diabetes should be relieved of infection as soon as possible.

3. Great harm is done by prolonged periods of observation because of the insidious manner in which infection progresses when associated with diabetes. When a surgical procedure is deemed necessary, it should be carried out immediately if acidosis is not present. Watchful waiting is dangerous.

4. Operative procedures in cases of diabetes should be just as complete and just as radical as in cases in which diabetes is not present but the same symptoms and signs are manifested.

5. There should be complete cooperation between the internist and the surgeon in the care of a patient who has diabetes and need of surgical treatment.

DISCUSSION

Dr. William C. Boeck, Los Angeles: Surgical intervention in the ear in cases of diabetes offers more of a problem than surgical intervention elsewhere in the body.

The cause of extension of the infection can be attributed only to inadequate surgical procedure; the area of infection has not been drained by the first operation, which should have been more extensive. There are such instances in the paper that has just been read.

It is not necessary to maintain the urine free from sugar in order to have satisfactory healing of the wound. Normal repair of the wound will take place with a decreasing amount of sugar in the urine even though the blood sugar level may be as high as 180 or 200 mg. per hundred cubic centimeters for four or five days after the operation. The reason a diabetic patient may prove a poor risk is not so much that his diabetes cannot be controlled as that arteriosclerosis comes earlier; death occurs most often from coronary thrombosis or uremia with arteriosclerotic kidneys, except in patients in the early and middle age groups.

I cannot refrain from encouraging the use at once of one of the drugs of the sulfanilamide group, preferably sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), whenever acute tonsillitis, acute pharyngitis or acute sinusitis occurs, in order to prevent the extension of the infection to the ear. By such treatment a London hospital reduced the aural complications from 14 to 2 per cent over a four or five year period.

Dr. M. C. Myerson, New York: As a rule, my associates and I use local anesthesia as most practical in cases of diabetes with aural involvement. One must appreciate that the course of toxic infection is much more rapid in a diabetic

than in a nondiabetic patient. Therefore, the rule that destruction of the bone occurs on the twelfth, fourteenth or sixteenth day in cases of mastoid disease does not hold in cases of diabetes. The clinician should not feel that he is breaking any rule if he operates on the fifth, sixth, seventh or eighth day, because it is not at all uncommon to find in a diabetic patient the same amount of destruction on the seventh or eighth day that one finds in a nondiabetic patient in the fourth week.

All know that diabetes has a masking effect on surgical disease. It is also recognized that the sulfanilamide group of drugs have a masking effect. It is much better to use sulfanilamide during the period of convalescence if one wants to use it.

DR. HARRY W. LYMAN, St. Louis: The first impression made on me by this paper is the contrast between the recovery of a diabetic patient with an aural infection now and in preinsulin days. Before insulin the operative mortality was high. The postoperative recovery of those that survived was almost undeterminable. The next thing that impressed me is one's tendency to think of these patients as having mastoid disease. One should be on guard at the beginning of the aural infection. Otitis media should be considered not just as otitis media but as potential mastoiditis of grave import.

DR. BERT E. HEMPSTEAD, Rochester, Minn.: We use local anesthesia in these cases as much as we can. We induce intratracheal anesthesia with gas and ether.

Newer Conceptions of Rhinootologic Meningitis. DR. JAMES G. DWYER, New York.

DISCUSSION

DR. LOUIS K. GUGGENHEIM, Los Angeles: It is important to decide whether one should use sulfanilamide and its derivatives for the prevention of intracranial complications or whether one should treat infections of the ear and sinuses in the traditional manner and await complications. It seems that much experience in the last year points to the value of drugs of the sulfanilamide group as a preventive in certain cases.

During the last few years I have come in contact with pediatricians and general practitioners who have made a fad of giving sulfanilamide and its derivatives for everything. A child has a slightly red throat and a little cough, and the mother brings it to the physician. One mother told me of such a case, in which the pediatrician promptly prescribed sulfathiazole. One should try to teach the pediatricians and the general practitioners that these drugs mask symptoms and that they should never be given unless a most careful examination is first made in order to recognize the possible complications.

DR. HAROLD FLETCHER, San Francisco: I cannot understand why people use a 10 per cent solution of sodium sulfathiazole in wounds, particularly in the mucous membrane. It is a very strong alkali, and one might as well put potassium hydroxide in a wound and expect it to do the same work. It is almost a caustic.

DR. ARTHUR JONES, Boise, Idaho: Seven patients treated with sulfathiazole were back in the hospital from ten days to two weeks after discharge with paradural or parasinal abscess. My colleague and I have finally persuaded pediatricians to stop giving this drug indiscriminately.

DR. JAMES G. DWYER, New York: In the average case in my own clinic, if the patient is doing well the sulfanilamide drugs are not used. If he is not doing well, he is brought into the hospital, where the blood count can be controlled. The treatment is not without danger.

Endocrine Basis for Otosclerosis. DR. J. MILTON ROBB, Detroit.

In conjunction with the work of Moehlig, Murphy and Reynolds, who have attempted to produce Paget's disease in dogs by the use of an anterior pituitary extract containing the growth-stimulating principle and solution of parathyroid

U. S. P., I studied the temporal bones of the dogs. These workers observed 4 dogs over an average of two hundred and thirty-one days, divided arbitrarily into three periods, one of one hundred, one of fifty-five and one of seventy-six days. Three dogs received combined daily injections of the extract containing the growth principle and the solution of parathyroid, and the fourth dog received only the parathyroid daily. The average total amount of parathyroid given was equivalent to 13,008 U. S. P. units.

A review of the literature revealed that this is the only experiment in which both solution of parathyroid and anterior pituitary extract containing the growth principle have been injected simultaneously and the temporal bones studied serially. From my observations in these experiments, it would seem that histologic changes occur in the temporal bone similar to those in human otosclerosis and that these changes were produced by the additional injection of a preparation containing the growth-stimulating substance.

DISCUSSION

DR. JAMES A. BABBITT, Philadelphia: Dr. Edward Rose reviewed the literature on endocrine glands for the last five years without finding grounds substantiating an endocrine basis for otosclerosis and questioned the potency of most anterior pituitary extracts. From a practical standpoint the present interest has stimulated the refinement of surgical procedure in the fenestration operations of Bárány, Sourdille, Holmgren and Lempert and in the round window block of Hughson. While receptive to the multitude of vitamin, estrogen and biochemical agents under experimentation, I should criticize the inexactitude of dosage and the enthusiastic trend toward hasty conclusions.

DR. LOUIS K. GUGGENHEIM, Los Angeles: Otosclerosis is a hereditary malady, which, however, being a bony process, is under the influence of a great many factors—a fact which offers the only hope of ever doing anything about the disease. It has been known for years that the process is more or less under the influence of the endocrine system, as are the bones, and there lies the possibility of doing something with the condition.

Laryngeal Tuberculosis: A Study of Five Hundred Cases of Pulmonary Tuberculosis, with a Résumé Based on Twenty-Eight Years' Experience. DR. JULIUS P. DWORETZKY, Liberty, N. Y., and DR. OTTO C. RISCH, New York.

Laryngeal tuberculosis is definitely on the decline. The incidence has dropped from 25.6 per cent in 1914 to 14.6 per cent in 1934, and to 3.6 per cent in 1941. This percentage is based on 500 cases for each year. The reasons for this are improved case findings, improved methods of diagnosis, earlier treatment and increased employment of collapse therapy.

Laryngeal tuberculosis is always secondary to pulmonary tuberculosis, and the larynx becomes infected chiefly in two ways: 1. By surface infection. This usually occurs in the bronchogenic type of pulmonary tuberculosis, in which the tubercle bacilli invade the larynx by penetrating the mucous membrane. 2. Through the blood stream. This form of invasion usually occurs in the hematogenous form of pulmonary tuberculosis, in which the larynx often becomes involved simultaneously with the lungs.

Tuberculosis of the larynx therefore occurs both in the bronchogenic and in the hematogenous type of pulmonary tuberculosis, but its clinical manifestations differ immensely in the two types, and unless the complication is properly recognized and differentiated, one will err greatly both in the prognosis and in the treatment. In attempting to make a differential diagnosis in these two types of laryngeal infection, it is well to remember the following distinguishing characteristics of the bronchogenic type:

1. The onset is insidious.

2. Examination of the chest often reveals physical signs indicative of pulmonary involvement.

3. The sputum is often positive for the tubercle bacilli, while in the hematogenous type the sputum is usually negative, especially in the beginning.

4. Roentgenograms of the chest are characteristic, showing infiltration of one or more of the lobes, often with cavitation.

5. The first symptom is usually hoarseness or irritation in the larynx, which necessitates frequent clearing of the throat.

6. The laryngeal lesion is usually limited to a portion of the larynx, most often to the posterior commissure or to one or both vocal cords.

7. The larynx is usually involved alone, and extension to other parts, such as the tonsils, pharynx or uvula, is rare.

8. The prognosis, as far as the larynx is concerned, is usually good, and it is particularly favorable when collapse therapy is successfully applied.

9. The laryngeal lesions usually require little treatment.

DISCUSSION

DR. M. C. MYERSON, New York: I should like to expose the myth that the early recognition of tuberculosis of the larynx is important in the end result in the individual case of tuberculosis of the larynx. A patient under treatment in a hospital can have a perfectly normal larynx today and then have a severely acute, destructive lesion in the larynx within twenty-four hours of the examination. As a matter of fact, he may have generalized disease in every part of the larynx, so that early recognition simply means early knowledge of that lesion. It does not mean that it has anything to do with the type and the ultimate outcome of the lesion.

A word about the percentage of cases: It is my belief that 25 per cent is much closer to the correct figure for 1941.

Because of the shortcomings of present diagnostic methods, any type of hematogenous lesion may be present in the larynx without its being discernible.

DR. JULIUS P. DWORETZKY, Liberty, N. Y.: My experience with the cautery in cases of the hematogenous type has been extremely unfortunate; most patients die. I found that they died not so much because of the laryngeal lesion as because the pulmonary lesion was so terrifically severe. I do not use the cautery in the cases of bronchogenic type with surface infections.

I shall have to differ with Dr. Myerson's statement that it is not important whether or not one diagnoses laryngeal tuberculosis early. I think it is very important. If one finds an early lesion one can advise the patient what to do.

Relation of Otolaryngology to Dental Pathology. DR. THOMAS E. CARMODY, Denver.

The artificial divisions that have been made in medical practice have not improved the situation for the patient. The multiplicity of organs involved and the possibility of error as to what part of the organ may be affected may confound the most astute diagnostician.

The interrelation of various lesions of the teeth, mouth, salivary glands, larynx and ear is discussed.

DISCUSSION

DR. JAMES G. DWYER, New York: Given a case of arthritis, the question is whether the cause is infection of a tooth, a tonsil, a sinus, the intestinal tract, the gallbladder or some other organ. That is the difficulty one encounters. There may be dozens of cases of pyorrhea, but unless there are pockets of pus arthritis will seldom result. Then there are cases of pus under pressure extending down to the teeth. Such a condition may also occur if tonsillectomy is incomplete and a piece of the tonsil tissue is left. Of the three—the teeth, the tonsils and the sinuses—I think the sinuses are most seldom the offender.

DR. D. C. JARVIS, Barre, Vt.: Looking in the mouth is the quickest method of estimating the calcium-phosphorus balance. First of all look inside the lower

front teeth. If there is calculus there, high calcium and low phosphorus are indicated. Notice the color of the mucous membrane of the gum around the upper front teeth. If it is pale the phosphorus is low and if it is red the phosphorus is high.

Injuries to the Nose in Children: Diagnosis and Treatment. Dr. Samuel Salinger, Chicago.

This article was published in the November 1941 issue of the Archives, pages 936-951.

DISCUSSION

Dr. Fred Z. Havens, Rochester, Minn.: I should like to reemphasize the necessity of removing any debris and of proper splinting. If these are neglected one is going to have disagreeable results.

Dr. John Mackenzie Brown, Los Angeles: I should like to ask Dr. Salinger a question. In a youngster from 6 to 10 years of age, one frequently finds a deformity of one side or the other that does not completely block the breathing. Have you found any difficulty in performing these operations at that age? Do you hesitate at all if the deformity is of sufficient proportion to interfere with the respiration on either side?

Dr. Samuel Salinger, Chicago: I do not advise doing a submucous resection on a child in whom the deviation is only moderate or is not associated with an external deformity. If the child can get enough air through even one side of his nose and the external deformity is not pronounced, I should not advise operation. The use of dental wax in the nose has been ideal in many cases. I have never seen anything like the usual accumulation of mucus or irritation, and for that reason I am enthusiastic about its use.

Atypical Facial Neuralgia. Dr. Robert C. Martin, San Francisco.

This article will appear in a later issue of the Archives.

DISCUSSION

Dr. David R. Higbee, San Diego, Calif.: The more I read about the sphenopalatine ganglion the more I am convinced that it has no relation to the sensation of pain. One must discriminate, however, between fibers which run along the course of the sympathetic and parasympathetic systems and which are not closely related to them.

Dr. Ben R. Dysart, Pasadena, Calif.: After much reading and rereading of this and other articles, I come back to derive what consolation I can from Dr. Martin's statement: "There is great confusion concerning the mechanism of the production of this head pain."

When possible one should find and remove the underlying cause of this neuralgia. The majority of patients show no visible pathologic condition. I formerly made a diagnostic irrigation of the sphenoid sinus in each case but rarely found any evidence of infection. A careful history is the most important help in diagnosis.

Many of the patients with this condition are allergic. In a case reported several years ago by Dr. Arbuckle three operations on the frontal sinus failed to relieve severe frontal pain. An allergic work-up and elimination of wheat stopped the pain completely. I have never felt safe in injecting alcohol into the sphenopalatine ganglion because of the occasional bad results that follow. If removal of the ganglion can be done safely, and Dr. Martin feels that it is not difficult, this procedure may prove to be the method of choice in treating severe forms of the condition.

Dr. Harry W. Lyman, St. Louis: The anatomic and physiologic discussion of this problem is one thing, and the clinical is another. I feel that this complicated

supply of nerve fibers in the head accounts for such a complicated clinical picture as atypical neuralgia presents. Dozens of types of headache may be included under this classification.

I reported a series of 6 cases in which neuralgia was relieved by surgical treatment of the sphenoid and ethmoid sinuses. In every one of these cases definite infection was present in the sphenoid or in the posterior ethmoid sinus or in both.

One is helping to solve this problem by classifying the cases of this condition. As to the removal of the ganglion, I have not attempted it yet. As long as the simpler methods give relief, at least in my hands, I feel that an operation should be reserved for those cases in which the pain cannot be controlled or relieved by such methods.

Dr. T. E. Walsh, St. Louis: I was much interested in Dr. Martin's paper. My associates and I have tried to give relief in these cases of neuralgia by making injections into the sphenopalatine ganglion, but I must admit we have not had very satisfactory results, except temporarily. In many cases we have not been able to find the source of the allergy.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 11-15, 1942.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.
Place: Hotel Dennis, Atlantic City, N. J. Time: June 8-9, 1942.

American Laryngological Association

President: Dr. T. E. Carmody, 277-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 25-27, 1942.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: June 1-3, 1942.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 28-29, 1942.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.